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DISEASES OF OCCUPATION

AND

VOCATIONAL HYGIENE

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To

GEORGE MILLER STERNBERG, M. D., LL. D.
A PIONEER IN PREVENTIVE MEDICINE IN THE UNITED STATES TO WHOM THE SENIOR EDITOR IS INDEBTED FOR AID AND ENCOURAGEMENT IN HIS WORK

AND

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PREFATORY

Historical Review

One of the most interesting and beneficient subdivisions of hygiene is a study of the relations of occupations to the health and longevity. The necessity of devoting special attention to this subject was shown long ago by observations made by Hippokrates and Galen that certain occupations and trades, even in those primitive periods, were dangerous to health. These and subsequent authors refer to their writings to occupational diseases of miners, bearer of burdens, messengers, sailors, soldiers, chemists and professional men, but the first systematic treatise on Diseases of Occupation was written by Professor Bernado Ramazzini of the University of Padua, in Italy. His Monograph "De Morbis Artificium Diatriba" published in 1700 was translated into English in 1705 and also into French in 1711. It awakened a deep interest in these countries and also in Germany.

Space will not permit to trace the rise and growth of domestic and handicraft industrial production and its influence upon the health of the operatives, which doubtless was very important when we recall the primitive workshops of the mediæval towns and cities. There is evidence to show that the factory system originated in the beginning of the sixteenth century, since we read of woolen cloth, linen factories, dye establishments, hat and soap factories, sugar refineries, etc., both in England and Germany, where large numbers of carders, spinners, weavers and other operatives were employed. The modern factory system probably started in the latter part of the eighteenth century. Stieda informs us that Germany prior to 1801 had about 20 factories, employing between 100 and 500 persons. It is quite natural that the first mechanical industries should have been devoted to the production of material for clothing.

The Spinning Jenny invented and perfected by High, Kay & Hargreaves in 1767 supplemented by Arkwright's spinning machine in 1771, and Cartwright's power loom patented in 1785 laid the foundation for the textile industry on a large scale. With the discovery of the steam engine and its application to factories in 1785 a tremendous rise in the mechanical and manufacturing industries occurred. This is shown by the fact that the importation of raw cotton into England increased from 5,000,000 lb. in 1775 to 273,249,653 in 1831 and also by an unusual demand for labor, especially apprentices and child labor to feed the machines.

With the development of the modern factory system of increased production, came also a concentration of the population in certain manufacturing

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districts, as witnessed by an increase of the population in the Lancashire District from 166,200 in 1700 to 1,336,854 in 1831. But most important of all, occupational diseases and accidents assumed more and more importance. It soon became apparent, in countries like England, France, Germany and Italy, that death was exacting a very heavy toll in many of the industries. Indeed the workmen in these countries began to believe that steam and speed had not improved their lot in life and spoke of their condition as one of slavery, and of their factories and workshops as "slaughter houses."

This was not an unreasonable conclusion, when it is recalled that in 1833 in factory towns like Manchester and elsewhere "the youthful population was physically worn out before manhood" and the average age of the laboring classes was only 22 years, as compared with 44 years among the higher classes. Even later there was a period when, with a general death rate for the whole of England of 22 per 1000, the death rate in the laboring districts was 36 out of every 1000 per annum.

In the interest of humanity, and as a matter of social and political economy, it became imperative to study the so-called dangerous trades to determine the sources and significance of the dangers and the possible means for the prevention and mitigation of the injurious effects. In the meantime Sir Robert Peel had caused the enactment of laws in England, as early as 1802, for the protection of the health and morals of apprentices and others employed in cotton and other mills and factories, which limited the hours of work to 12 hours a day, and made provisions for general cleanliness and also for periodical inspections by a Justice of the Peace and a Clergyman. In 1816 and in 1833 the English Parliament appointed a commission, to inquire into the condition of factories, which resulted in 1833 in placing an age limit on child labor, in the appointment of four regular factory inspectors and in the enactment of special rules for the white-lead industry and bake houses, etc. In 1844 the employment of children under 8 years of age in factories was forbidden and female labor was restricted. In 1847 the hours of labor for women and children were limited to 10 hours a day. In 1854 a law requiring machinery guards was enacted. In 1864 certain occupations were declared dangerous and in 1867 a law compelling the installation of appliances for the removal of dust was enacted. An Act of 1878 permitted children to be employed at the age of 10 years, but excluded them from work in certain of the processes in the white-lead industry. The Act of 1891 raised the age limit to 11, which has since been raised to 13 and 14. This Act also included workshops, under the provisions of all factory acts and placed the supervision of the sanitary condition of the workshops in the hands of the local sanitary authorities. In 1901 increased powers and duties were conferred upon medical officers of health. In 1897 the Workmens' Compensation Act was passed, which was amended in 1900 and again in 1906. Other countries followed the lead of England. In 1810 the French Government issued a decree relating to "establissements dangereaux insalubres et incom-

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modes" and in 1839 the Academie des Sciences morales et politiques de France" and subsequently Prussia, Bavaria and other German states directed similar investigations.

In the prevention of the fundamental cause of poverty and distress the medical profession has been a help mate to religion. Men who come into daily contact with sickness and sorrow cannot fail to experience a deep sympathy for their fellow men, which is all the more profound when they realize that many of the diseases are preventable and hence much of human suffering cruelly unnecessary. As a result, a special department of Social Medicine has been created with a most complete and satisfactory literature of its own. As early as 1822 C. Turner Thackrah, of Leeds, wrote a monograph on the effects of the Arts, Trades, and Professions, and of civic States and Habits of living, on Health and Longevity." In the same year Patissier wrote his "Traité des Maladies des Artisans" and in 1845, Halford, of Germany, published a very important work on occupational diseases. Since which time numerous contributions on the causes, mortality and prevention of occupational diseases and the hygiene of occupations have appeared. German authors, under the leadership of Theodor Weyl, in 1897 issued a volume of over 1200 pages, and English authors under the Editorship of Dr. (now Sir Thomas) Oliver devote 891 pages to "Dangerous Trades: The Historical, Social and Legal aspects of Industrial Occupations as affecting Health."

These monumental volumes are especially mentioned because the contributors were recognized experts on the various subjects and hence their conclusions should carry great weight. This in no way sould be construed as underrating the value of pioneer workers like Thackrah, Halford, Arlidge, Eulenberg, Hirt, Patissier, Layet, Oldendorff, Roth, Albrecht, Sommerfeld, Dammer and a host of others, whose writings will always remain monuments of careful study, accurate observation, great industry and faithful devotion to a noble cause.

These individual studies, and numerous governmental investigations, have supplied more accurate information concerning the causes of disease and accident—hazards in different occupations and have provided forceful argument for protective legislation and education. Much has been accomplished, in the way of industrial and social betterment, by the establishment of industrial insurance, the erection of sanitary homes for wage earners, and the enactment of workmen's compensation laws in case of accident or injury, from occupational diseases, contracted in the line of duty. In the way of educational methods, industrial museums of safety have been established in 14 European cities. The Berlin Museum established in 1904 and the Vienna Museum founded in 1909 are typical examples of what may be done in illustrating the cause and prevention of occupational diseases and accidents. In the Berlin Museum, located at Charlottenburg, every safety appliance which inventive genius has devised can be seen in practical operation. The

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different labor unions appear to profit immensely by the special lectures and demonstrations which are given on Sundays or, upon request, at any convenient time, by men formerly employed in "Dangerous Trades." Apart from safety devices for machinery and appliances for the removal of dust and injurious gases, all improved methods calculated to diminish danger, as for example, in the manufacture of white lead, etc., are illustrated by models and descriptive text, printed leaflets being distributed free of charge. Here to, may be seen the latest and best types of respirators, wire masks, goggles, illuminating appliances, first-aid kits, safety working suits, etc. Only meritorious objects are displayed, and they are replaced by newer and more satisfactory types. One of the most interesting collections consists of a series of raw materials, a series of bottles containing dust evolved from such material, a series of photographs showing the microscopical character of this dust, and last, but not least, anatomical specimens and microscopical slides, showing the effect of dust upon the respiratory organs of the human subject. Models, plans and photographs of sanitary homes for wage earners with exterior and interior decorations have a prominent place in the exhibit. The display of food stuff, and data relating to their nutritive and economic value together with instructive pamphlets, such as "How to keep well and capacitated for work," "Good food at reasonable cost," "The Alcohol Question," "The Social Evil," etc., form part of this interesting exhibition. In 1906 the first International Congress on Occupational Diseases was held at Milan, which city has also the proud distinction of establishing the first, and so far the only hospital and clinic for the treatment, study and prevention of Occupational Diseases. A report of the activities of this clinic by its distinguished Director Professor L. Devoto will be found on page 765. These measures and the establishments of laboratories in connection with the universities for the development of industrial hygiene, and the formation of Research Institutes for Industrial Hygiene, such as the one at Frankfort on the Main, in 1910, have been most beneficient in the interest of wage earners.

Progress of Industrial Hygiene in the United States

As in Europe so in this country the mechanical industries had their origin in the textile industry. According to Wright cited by Price, the first spinning jenny in this country was exhibited in Philadelphia in 1775. The first cotton factory to apply Arkwright's invention was started with three cards and 72 spindles in Rhode Island in 1790, by Samuel Slater of England. It is reported, however, by a writer in the American Museum for July, 1790, that a man in South Carolina, completed and had in operation on the Santee, ginning, carding and other machines driven by water and also spinning machines with 84 spindles each. Waltham, Mass., has the credit of having set up the first power loom in 1814, and of having established for the first time in history, a factory in which all the processes involved in the manu-

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facture of goods from the raw material to the finished product were carried on in one establishment.

The wonderful progress in the cotton textile industry in the United States is shown by the increase of spindles from 4500 in 1805 to 1,246,703 in 1831. As in England so in this country the application of machinery resulted in the exploitation of child labor. It cannot be said, that we followed the example of England to the extent of trafficking with the poor law officials, who according to Price sold the children of paupers in a form not different from the methods of ancient and modern slave dealers. It is reported, however, by Price that the first cotton mill established in 1790 in Rhode Island began to work with four spinners and carders, but five children were soon added, whose ages ranged from 7 to 12 years, and in 1831 the number of children working in the cotton mills of that state was almost one-half of the total number of employees.

According to Miss S. S. Whittelsey's essay on Massachusetts labor legislation, the subject of Child Labor received attention in 1836. The Act provided for at least 3 months schooling during the working year for every child employed under the age of 15, which was amended in 1842 by fixing a 10-hour working day for children under 14 years. Similar legislation was enacted in Connecticut in 1842, in Maine in 1847, and after 20 years agitation also in Pennsylvania in 1848. Massachusetts was also the first state to enact a law in 1852 as regards safety of steam machines and by an Act of 1870 also required supervision of steam boilers, but it was not until 1877 that this state enacted a law setting forth certain requirements for the removal of dust. Since this time nearly all of the states have enacted some form of factory legislation, but even at the present time only 22 states have provisions for protection against injurious dust, 15 require notification of certain occupational diseases, 12 states have legislated against defective lighting and 11 on the subject of temperature and humidity. There is a distinct need for a definite standardization of factories with reference to construction, fire protection, light, air, space, ventilation, safety against accident and general sanitation. A lack of appreciation of the importance of enforcing existing legislation is clearly evinced by the fact that even the State of Massachusetts made no provisions until 1888 for a separate department of factory inspection, the duties having been performed up to that time, by truant officers and members of the police force.

In the mean time the Department of Labor has published a number of interesting studies, notably the essays on "Employer and Employees" by D. H. Olmsted and S. D. Fessenden, in Bulletin 1; "The Sweat System" by Henry White, Bulletin 4; "The Inspection of Factories and Workshops in the United States" by William F. Willoughby, Bulletin 12; "The Production of Paper and Pulp," Bulletin 23; "Protection of Workmen in their Employment" by S. D. Fessenden, Bulletin 26 and also numerous articles on foreign

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labor laws. These writings have done much to stimulate the enactment of labor and factory laws.

In 1902 Dr. Kober, upon request of the Hon. Carroll D. Wright, Commissioner of the Bureau of Labor recommended his former student Dr. C. F. W. Doehring for the purpose of making an investigation into the manufacture of white lead, paint, etc. The manufacture of linseed-oil, oilcloth and linoleum, and the manufacture of tallow, fertilizers, etc. The results of this investigation were published under the title of "Factory Sanitation and Labor Protection" in Bulletin 44, January, 1903. In 1905 the State Board of Health of Massachusetts, being the first State in the Union to recognize the fact that sanitary inspection of factories was essentially a public-health matter, submitted a brief report on "The Conditions Affecting the Health and Safety of Employees in Factories and Other Establishments." This report was supplemented in 1907 by a more exhaustive report and also by an exhibition of 90 photographs. This was in fact the first exhibit in America relating to occupational diseases. The photographs, based upon a study of dusty trades in Massachusetts, by Dr. William C. Hanson, together with the charts, and a collection of dust and other materials, demonstrated in a most effective manner that many of the insanitary surroundings and injurious factors in the leading industries are due to neglect of perfect ventilation, and hence are to a great extent avoidable. In September, 1907, during the International Congress on Hygiene at Berlin the Senior Editor met Dr. E. J. Neisser, who had just completed an International Review of Industrial Hygiene covering a volume of 352 printed pages. Dr. Neisser deplored his inability to present an extended review of the work accomplished in the United States, since with the exception of one or two states, no data concerning factory sanitation were available.

Realizing, as Chairman of the Committee on Social Betterment of the President Homes Commission, the importance of the subject, not only to wage earners, but to all interested in the conditions under which our fellow men and women live and work, Dr. Kober in February, 1908, submitted a report on Industrial and Personal Hygiene covering 175 pages. This was followed in December, 1908, by a Report of the Committee on Social Betterment covering 281 pages. It was hoped that this somewhat hasty study of the causes of sickness, and the means of promoting industrial efficiency and earning power, would direct attention to the need of a more critical study, and of remedial legislation in this Country. This hope, thanks to the medical and lay press and the interest aroused among officials and social workers, has been realized. In November, 1908 and May, 1909, Dr. Frederick L. Hoffman's valuable monographs on "The Mortality from Consumption in Dusty Trades," were published by the U. S. Bureau of Labor. In January, 1910, the Epoch-making report by Dr. John B. Andrews on "Phosphorous Poisoning, in the Match Industry in the United States," appeared in Bulletin 86 of the same Bureau. In 1910 the "Illinois State Commission on Occupational

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Diseases" began its labors and in 1911 published a large number of specific instances of industrial poisoning contracted by wage earners in the course of their employment. In June, 1910, the "First National Conference on Occupational Diseases" was held at Chicago, and in the memorial to President Taft it was estimated that 13,000,000 cases of sickness, involving an economic loss of nearly three-fourths of a billion dollars, occurred annually among the artisans and craftsmen of the United States. In July, 1911, the Bureau of Labor published in Bulletin 95 the result of an intensive study by Dr. Alice Hamilton, which disclosed 388 cases of lead poisoning, of which 16 were fatal in the white lead and lead oxide industries in the United States from January 1, 1910 to April 30, 1911. The same bulletin also contained a report by Dr. John B. Andrews on 60 deaths from industrial lead poisoning actually reported in New York in 1909 and 1910. The report of lead poisoning in potteries, tile works and porcelain-enameled sanitary ware factories by Dr. Alice Hamilton and her admirable monograph on the hygiene of painters were published by the Bureau of Labor statistics, respectively August 7, 1912 and May 13, 1913. These were followed by a report on lead poisoning in the smelting and refining of lead, February 17, 1914. Dr. William C. Hanson's monograph on the dangers to workers from dust and fumes and methods of protection illustrated with 62 photographs, was published by the same Bureau, August 12, 1913. Apart from Illinois, the States of New York, Wisconsin and Missouri have created special commissions for the study and prevention of occupational diseases, and important investigations have been made by Drs. J. H. Lloyd; George M. Price; Mrs. Lindon W. Bates; Dr. C. T. Graham Rogers; Dr. D. L. Edsall; E. E. Pratt; E. R. Hayhurst and numerous other official and private investigators. The 6th International Congress for the Prevention of Tuberculosis held in Washington in the fall of 1908, and the 15th International Congress on Hygiene held in the same city in September, 1912, with their excellent exhibits illustrating the dangers of numerous occupations, stimulated a profound interest in the subject of industrial hygiene, and the National Association of Labor Legislation organized in 1908 and numerous independent organizations have accomplished excellent work in the way of investigation and legislation. In 1911 the American Museum of Safety was incorporated under a special charter from the legislature of New York. It is located in the City of New York and is devoted "to the safety, health and welfare of industrial workers and the technique and science of industry." Dr. Wm. H. Tolman is the Director of the Museum and Dr. N. E. Ditman is in charge of the department of industrial hygiene. For the purpose of stimulating invention and the installation of safety devices and the promotion of sanitation in all industries the Museum has awarded a number of medals.

In 1911 the "National Safety Council" was organized and later created a section on Industrial Hygiene which held its first session in connection with the Fourth Annual Congress of the National Safety Council at Philadelphia

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in October, 1915. The American Public Health Association also created a section on Industrial Hygiene, which held its first meeting at Rochester, N. Y., in September, 1915.

In 1912 the U. S. Public Health Service created a Division on Industrial Hygiene with Dr. J. W. Schereschewsky in charge and in 1915 established a research laboratory in the city of Pittsburgh, Pa. In the mean time excellent field work has been carried on in some of the mining districts and in various industries. There is every reason to believe that the investigations of this division will accomplish much towards a standardization of industrial plants. as regards sanitation, equipment and the prevention of accidents and also in the matter of medical supervision and medical relief of the personnel.

In 1915 and 1916 the U. S. Government assembled an excellent exhibit of safety devices in the National Museum in Washington. This exhibit, beginning May 1, 1916, will become a railway travelling exhibit and visit every important industrial city in the United States.

While the out-patient department of the Massachusetts General Hospital in Boston and the Clinics connected with some of the University hospitals in New York City and elsewhere, have given more attention to the study of occupational diseases during the past decade, it was not until 1915, that the first Clinic exclusively devoted to occupational diseases was established in this country by authority of Dr. S. S. Goldwater, in connection with the Health Department of the City of New York. It is hoped that this example may be followed by other American cities.

In 1912 Miss Josephine Goldmark of New York published a very comprehensive Study of Fatigue in its industrial aspects.

Dr. W. Gilman Thompson was one of the first among American Physicians to appreciate the importance of occupational diseases from the medical, social and economic point of view. Apart from organizing in New York City, in November, 1911, an informal committee "for the purpose of studying occupational diseases, and especially for coöperating with the State Labor Bureau, in furtherance of its investigations," he has made a number of valuable contributions to the literature, the latest being his work on "Occupational Diseases," which appeared in June, 1914. Dr. Geo. M. Price's latest contribution appeared in 1914, in the form of a book of 574 pages, entitled "The Modern Factory."

In justice to the publishers of the present volume, it should be stated that the Senior Editor declined their request to prepare or edit a book on the "Diseases of Occupation and Vocational Hygiene" in the early part of 1911, because the scarcity of statistical data in reference to disease and accident-hazards in the various occupations of this country, had compelled him in his former writings to get most of his facts from Europe and the State of Massachusetts.

He entertained the hope, that with a general awakening in this country, and the publication of the transactions of Section IV of the XV Inter-

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national Congress of Hygiene and Demography, devoted to the hygiene of occupations, over which he was called to preside, additional material on the diseases of occupation, as they occur in various states of the Union, and in foreign lands, would be available. This hope has not been vain. Then in addition to the transactions of Section IV, the publication of the latest mortality statistics by the U. S. Census Bureau, the collection of industrial insurance statistics by the Metropolitan Insurance Company, and the issuance of monographs covering special investigations made in the States, notably by Dr. E. R. Hayhurst in Ohio, in 1915, has enabled us to write more authoritatively than ever before on disease and accident-hazards of the American worker. Special acknowledgment is made to Drs. Henry H. Hazen and John D. Hird, and to Professor Charles E. Munroe, and Mr. Charles H. Verrill and Mr. R. C. Lappin for valuable assistance.

THE EDITORS.

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FOREWORD

The constant aim of the Editors has been the presentation of the basic data concerning the diseases of occupation in such a way as to render them available not only to physicians, but also to employers, employees, efficiency experts, public health officials and legislators; for it is only as a knowledge of the character, gravity, causes and prevention of these diseases is diffused that corrective and preventive action can be expected. The subject matter here presented is grouped, therefore, in three parts.

Part I deals with the Specific and Systemic Diseases of Occupation. It also treats of Fatigue and the Neuroses. It is written by specialists of international reputation and adapted to the needs of medical and legal experts, medical examiners, insurance examiners, practising physicians, teachers, students and others desiring accurate scientific information relating to the pathology, symptomatology and treatment of diseases of occupation.

Part II deals with the Causation and Prevention of Occupational Diseases and Accidents. While it is also of interest to physicians, lawyers and efficiency experts, its chief interest should be with employers, employees, public health officials, nurses and social workers, legislators, and others actively interested in the prevention of avoidable occupational hazards. This part of the book also contains a list of industrial poisons and of industries and processes in which poisoning may occur, and also tables of occupational mortality and morbidity statistics, arranged in alphabetical order for convenient reference.

Part III is intended to be of service to those who may be called upon to investigate in the shop, the factory, in the dispensary and hospital the relations of occupation to disability and disease. This portion has been framed, too, in the hope that it may stimulate the teachers of the coming generation of physicians to impart to their pupils a better knowledge of occupational diseases and their prevention, which after all is of more economic importance than their cure.

The Editors have endeavored to make the book a safe and convenient guide to all who may be interested in the study of occupational diseases. They ask, therefore, the kind indulgence of the reader who may discover in it errors or shortcomings and will be glad to have such defects brought to their attention.

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PART I

SPECIFIC AND SYSTEMIC DISEASES OF OCCUPATION

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DIVISION I

Specific Occupational Diseases

CHAPTER I

OCCUPATIONAL INTOXICATIONS

SECTION I

ARSENIC POISONING

BY T. M. LEGGE, M. D., London, England.

Industrial arsenic poisoning occurs in two distinct ways: (1) from the dust of salts of arsenic, through inhalation or contact; and (2) from arseniuretted hydrogen gas.

The principal poisonous salt is white arsenic, or arsenious acid (As_2O_3).

Pure metallic arsenic (As) is considered to be innocuous.

Arsenic: How Obtained; Its Important Uses.—In order to obtain white arsenic (arsenious acid) the ore is roasted and the arsenic so volatilized is collected in flues and chambers. This so-called "arsenic soot," in the collection of which elaborate precautions in the shape of overalls and respirators are necessary to guard against the effects on the skin, is again submitted to heat in a refining furnace and the fumes again deposited in flues as white as a "hound's tooth." Subsequently, the material is ground and packed in barrels usually by automatic arrangements preventing dust. Use of emerald, Paris or Schweinfurt green (aceto-arsenite of copper, $Cu(C_2H_3O_2)_2Cu_3As_2O_6$) depends on its value as an insecticide in preserving fruit trees. In its manufacture the workers run little risk until the precipitate of emerald green (made by mixing solutions of arsenite of soda with sulphate of copper) is dried. All subsequent processes of sifting and packing, unless carried on in closed-in apparatus from start to finish, cause the greatest distress to the workers, owing to the very irritant nature of the excessively fine and light dust which "flies" readily.

Use of Scheele's green (arsenite of copper) in the preparation of artificial flowers and wall papers has only a historical interest because aniline colors have almost entirely taken the place of arsenic in their manufacture, and a host of other articles (notably children's toys), as a consequence of the injury traced to their action several years ago. When arsenic colors were used, as much as from 1 to 50 or 60 grains was found per square foot in different samples of wall papers examined for a committee of the Medical Society of London. The progress made since then is well illustrated in a Report of the United States Department of Agriculture in 1904, following on the law

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passed in 1900 by the State Legislature of Massachusetts limiting the amount of arsenic in wall papers and woven fabrics, other than dress goods, to 0.10 grain per square yard, and in dress goods and articles of dress to 0.01 grain per square yard. Thus, of 537 samples of paper examined only four contained more than 0.10 grain per square yard and 90 per cent. of them contained less than 0.046 grain. The importance of the matter in relation to wall papers is that symptoms of coryza and conjunctivitis, gastro-intestinal catarrh and obscure nervous symptoms, all suggesting arsenical poisoning, are found to be associated with residence in rooms the wall papers of which contain arsenic. How the arsenic came to be disengaged from such papers remained a mystery until it was discovered that certain moulds and fungi (notably penicillium brevicaule) while growing upon the adhesive paste split up the arsenic compounds, liberating them in the form of a gas, di-ethyl-arsine ($AsHET_2$). Thus technical improvement in processes due to progress in chemical knowledge is shown in the case of arsenic colors. In some of the aniline colors themselves, as of fuchsin red, the use of arsenious acid in the original method of manufacture has been dispensed with by adoption of the nitrobenzene process.

In its effects on the workers by reason of the lightness and readiness of the dust to "fly," manufacture of sheep-dip—a mixture of solutions containing white arsenic, caustic potash or soda to which flowers of sulphur are added—closely resembles that of emerald green.

Other important uses of arsenic are of the oxide as a preservative of hides, skins and furs, and of red orpiment (As_2S_2) as a depilatory in tanyards. After the liming process the hides are pasted (on the flesh side) with a mixture of the red arsenic and lime. About 5 lb. of arsenic and a bushel of lime are mixed to a thin paste with water in a tub and the mixture applied to the hides with a piece of sacking tied to the end of a pole. Each hide, after pasting, is folded once, and the hides are then piled in heaps for about 12 hours, when the depilatory action is sufficiently advanced to allow of removal of the hair by scraping the hair side of the hides on the beam. In 1912 three men employed at the London docks suffered from sharp attacks of sore throat and diarrhea as a result of unpacking bird skins. From seven of the boxes it was stated that 16 lb. of arsenious acid were recovered.

Salts of Arsenic.—In the form of the salts arsenic acts as a local irritant or escharotic on such portions of the skin or mucous membrane as it may alight, whereas absorbed as a gas it acts hemolytically, destroying the red blood corpuscles and throwing the hemoglobin into the blood stream.

If poisons are described as (1) having a superficial action on the skin, (2) being absorbed by the blood, changing its constitution, and (3) having an internal, remote effect after absorption on organs or tissues such as the heart or nervous system, then arsenic, in a way in which no other substance does, displays all three modes of action.

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Symptoms.—The symptoms caused by dust of salts of arsenic show themselves on the skin, on the mucous membrane of the upper air passages, and in the gastro-intestinal tract. Dust alighting and remaining on the skin, especially where there are folds as around the nose and mouth, or where surfaces are moist as in the armpits or on the scrotum, sets up an acneiform eruption or an eczematous condition which, if not treated, will lead to extensive ulceration. Indeed the discomfort produced by this so-called "arsenic pock" is very great. On the other hand, a slow undermining of the health such as is commonly caused by lead poisoning is not evident. Associated with the skin eruption are conjunctivitis with œdema of the eyelids, coryza, injection with dryness and soreness of the throat, hoarseness (a very noticeable symptom), and depression. In severe cases the gastric symptoms become pronounced—vomiting, abdominal cramp, and purging. In former years workers were known to be sometimes so seriously collapsed as the result of the cramps as to require removal to their homes in cabs.

Peripheral neuritis showing itself in paræsthesia and anæsthesia ("pins and needles" feeling) is much commoner in my experience than paralysis of the muscles of the extremities.

Brown pigmentation of the skin is present in those who have worked for years in contact with arsenical dust. In slight cases it is seen best on the upper and lower eyelids, round the temples, on the neck, the areolæ of the nipples and in the folds of the axillæ, while in marked cases there may be an intense bronzing of the chest, abdomen and back.

The most characteristic lesion produced on the upper air passages is perforation of the septum of the nose varying in extent from a circular hole ⅛ to 1 in. in diameter. Perforation may be complete in a month from the time of commencement of work. The anterior and lower margins of the cartilage, and the bones to which they are attached, never become involved in the ulcerative process so that deformity is absent, thus distinguishing the condition easily from that due to syphilis. Once the perforation is complete no further inconvenience is felt; not a few of the workers are ignorant of the existence of the condition.

Sir Jonathan Hutchinson has laid stress on what he believes to be relatively frequent occurrence of cancer in those who have taken arsenic for long periods. A list of 31 published cases collected by Pye-Smith includes two in which the persons had been employed in a sheep-dip factory.

The following table shows the number of cases of arsenical poisoning (other than those due to arseniuretted hydrogen) reported to the Chief Inspector of Factories, London, during the years 1900–1913 inclusive and their distribution according to industry:

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Industry	Total cases
Manufacture of emerald green	46
Extraction of arsenic	8
Manufacture of sheep-dip	5
Paint and color works	4
Chemical works	3
Smelting of metals (lead, copper)	3
Sorting bird skins	3
Wall paper manufacture	2
Shot making	1
Scraping paint off ship	1
Tanning	1
Unloading white arsenic	1
Indefinite	8
Total	86

The symptoms described are those which inevitably result from the manufacture of, or contact with, the dust of salts of arsenic where hand labor is relied upon, even when protection is sought by the usual precautions of the wearing of overalls and respirators, locally applied exhaust ventilation, washing and bath accommodation and periodical medical examination. Fortunately progress in adoption of automatic methods involving no handling of the materials in a dusty condition has robbed the emerald green industry of danger. Thus, in a large factory where this has been achieved I examined 25 workers in 1900 of whom 20 had worked for 3 months or less (a short duration of employment generally signifies an unpleasant occupation with unskilled labor ignorant of the risk) and found only two free from signs of the discomfort and injury caused. As late as the year 1908 out of an average of 33 workers examined weekly 5.4 per cent. had to be suspended, as compared with 0.6 per cent. in 1913, the weekly average exposed to risk having been reduced to 14, owing to the improvements effected in the process. The dried material is here placed in an enclosed hopper connected with a powerful exhaust fan. When the door is shut the material is tipped over into a receptacle from which it is elevated into a sieve (also under negative pressure) and from there again passed automatically by means of a worm into the packing hopper and so into the tin canisters. Only as the green falls into the cans is there possible exposure to dust, and the man placing the cans in position is completely protected by the exhaust draught immediately behind them. No dust is visible from start to finish—a condition very different from that of a few years ago when the effect of the dust on the eyes and nose made one glad to get out of the atmosphere after 5 minutes. Splendid washing accommodation and douche baths now make the conditions wholly satisfactory. At the same time the wise precaution of periodic medical examination at weekly intervals has not been relaxed.

In the process of de-arsenicating sulphuric acid a large alkali works have issued the following notice of precautions to be observed by their workmen:

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ARSENIC RECOVERY PROCESS

Whereas Arsenic is a powerful Poison, it is very important that the greatest possible care should be taken by all who are engaged in its manufacture or brought in contact with it. The Officials and Workmen are, therefore, requested to strictly observe the following Rules, and generally to take every precaution to prevent Arsenical Poisoning:

1. In the process of De-arsenicating Vitriol, care must be taken to maintain an in-draught in the pipes and apparatus, to prevent the escape of Arsenical Fumes, and should an escape be observed, the Brine (or Hydrochloric Acid) should be at once shut off until the defect is remedied.

2. In the Precipitating, Drying and Packing operations, a clean Respirator (Grell's improved pattern) must always be used. A clean one must be obtained every morning from the Laboratory, and the one used the previous day returned to the Laboratory to be cleansed and prepared for use the next day.

3. Every workman engaged in this process shall be provided by the Company with a pair of India-rubber Gloves and a suit of Clothes, which he is to put on in the morning and remove on leaving the works at night, and shall take a bath before putting on his ordinary clothing. These special clothes to be washed by the Company and supplied clean to the workman once a week. Workmen must thoroughly wash their hands before taking food in the Works.

4. The buildings in which the manufacture of Arsenic is carried on must be well ventilated to remove all fumes of Chloride of Arsenic or dust of Arsenious Acid from the atmosphere.

5. Any workman having cuts or abrasions on the skin will not be permitted to work in the Arsenical Department until such wounds have quite healed.

6. Any workman showing the slightest signs of Arsenical Poisoning must be examined by a Doctor and undergo medical treatment.

Arseniuretted hydrogen gas, the extraordinary subtle poisoning, occurs mainly in the chemical metalliferous industries, but occasionally isolated cases will naturally occur in any chemical action of acid on metal when either one or both are arseniferous. The workman, therefore, absorbs the poison in operations in which as a rule the possibility of poisoning is not so much as imagined. Fortunately this form of poisoning is rare. Such cases as have come to my knowledge have been frequently due to work carried on in confined spaces or where the means for the removal of the gases evolved were inadequate.

The following table shows the number of cases reported by medical practitioners to the Chief Inspector of Factories in the years 1900–1913 and the industries in which they occurred:

Industry	Cases
Chemical works	20
Galvanizing works	7
Bullion refining	2
Bronzing of art metal	2
Paper manufacture	1
Tin-plate works	1
Total	33

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Professor John Glaister of Glasgow in his interesting monograph on this form of poisoning has classified 120 cases as follows:

I.	Chemical operations in laboratories	8
	(*a*) Operations with known arseniferous materials	1
	(*b*) Operations with unknown arseniferous materials	14
	(*c*) Operation not known	7
II.	Trade processes	73
III.	Military ballooning	16
IV.	Domestic environment (wall papers)	6
V.	Causes not known	2
	Total	120

The professions and occupations exposed to risk are classified thus:

I. Professional chemists: (*a*) medico-legal analyses in cases of arsenical poisoning; (*b*) in preparation of AsH_3 for demonstration purposes; (*c*) researches in aniline colors and in arsenical compounds.

II. Physicists and physiologists, from inhalation of arseniferous hydrogen in experiments on the voice or as to pulmonary capacity, or other like experiments.

III. Workers in aniline colors, from preparation of arsenic acid (a process now obsolete).

IV. Chemical workers: (*a*) manufacture of iron sulphate; (*b*) manufacture of zinc chloride and sulphate; (*c*) zinc smelting; (*d*) roasting and extraction of mineral ores; (*e*) manufacture of bleaching powder (calcium hypochlorite); (*f*) manufacture of soda sulphate by treatment of soda-lye with arseniferous sulphuric acid.

V. Ballooning for military and other purposes.

VI. Plumbers in brazing operations.

VII. Galvanizers.

The essential factor for the production of arseniuretted hydrogen gas is the presence of nascent hydrogen in solutions containing arsenic; hence its occurrence when hydrogen is generated from zinc, tin, copper, or iron, and sulphuric or hydrochloric acid when either or both show a proportion of arsenic. These substances and the acids, unless the sulphuric acid has been prepared from sublimed sulphur or has undergone a de-arsenicating process, are always more or less contaminated. Samples of sulphuric acid have been found to contain proportions of arsenious acid varying from as high as 0.045 to 0.140 per cent. and hydrochloric acid from 0.0014 to 0.691 per cent. Some of the cases among chemists, as, for instance, of Gmelin who discovered the gas, have followed the mere sniffing to detect its garlicky smell.

Symptoms.—The symptoms set in generally a few hours after inhalation of arseniuretted hydrogen gas, with nausea and sickness quickly followed by almost continuous vomiting. Jaundice of the skin and conjunctivæ comes on in the course of 48 hours and assumes in all but very slight cases an intense coppery hue; there is pain in the region of the liver, and hemoglobinuria or hematuria, and in the severest cases suppression of urine. All these symp-

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toms are referable to the hemolytic action of the gas upon the red blood cells which are thereby rendered incapable of supplying the coloring matter necessary for the internal respiration of the organs and so cause a condition of tissue suffocation. The functions of the liver and kidneys break down in the effort to eliminate the products of the disintegrated corpuscles. The characteristic jaundice is explained far more readily on the supposition that the dissolved hemoglobin circulating in the blood impregnates the tissues and thus communicates to them the peculiar color than that the liver, as a consequence of the abnormal condition of the blood reaching it, secretes an abnormal bile rich in bile pigment. The jaundice is, therefore, hematogenous and not hepatogenous.

After the discoloration of the skin the most characteristic signs post-mortem are seen in the kidneys, which are described as brown, chocolate brown, or brownish black; there is enlargement and the tubules are full of casts of broken-down corpuscles.

Several of the cases enumerated in the list of reported cases occurred in groups. Reference to some of them will serve to illustrate the circumstances under which nascent hydrogen was generated in arseniferous solutions and the kind of precautions which should be carried out in chemical works where there is risk of evolution of the gas. In some instances knowledge of them only slowly filtered through in consequence of the bizarre symptoms of jaundice suggesting yellow fever, acute yellow atrophy of the liver, or acute phosphorus poisoning. Doubtless some cases are never recognized.

In a works for the manufacture of bleaching powder a man engaged in the cleaning out of a Weldon chlorine still at the usual three weekly period was reported as suffering from "constant vomiting, coppery jaundice, lividity of the body, the mucous membrane of the lips almost black, suppression of urine—what little was drawn off consisting almost of blood." He died 6 days afterward. It transpired that a fellow-workman was affected in the same way though less severely, and that a few months previously an othe man had died after a similar cleaning operation. Of such stills there are throughout the country several thousands, and as no other cases were recorded there had obviously been unusual circumstances at work. The still in question differed from most similar stills in not having a trap-door at the base through which the residue could be discharged outside. Instead the residue was placed in a bucket and drawn out through the manhole above. The Weldon mud—acid calcium manganite—is run into the still which is then filled to a height of 2 ft. with hydrochloric acid. After the chlorine has been evolved the manganese chloride liquor is withdrawn, but a certain amount of residue remains behind. A probable source for the production of the gas lay in the hydrochloric acid made on the premises from pyrites. The deceased had used a new galvanized iron bucket and iron shovel for the removal of the residue. A sample of the liquid containing the deposit was found to contain 0.45 per cent. of arsenic acid and the hydrochloric acid used 0.292

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per cent. of arsenious acid. This the analyst described as "highly arsenical." It evolved, when iron and zinc was placed in it, arseniuretted hydrogen rapidly and abundantly. This acid I find to be very much more arsenical than the crude acids of commerce. Similar he said that to immerse zinc or iron implements in the liquid containing the deposit in an imperfectly ventilated still would involve great risk to life. In 11 oz. of the viscera which had been preserved in formaldehyde (equivalent to about 24 oz. of fresh viscera) 1/350 grain (0.0028) arsenicum was found and on this the analyst reported that "assuming the arsenic was uniformly distributed through the body, and that the deceased was a man of average height, the whole body would have contained 0.28 grain (rather more than a quarter of a grain) arsenicum. One and a half grains of arsenicum in the form of white arsenic taken by the mouth, has caused the death of an adult, and less than half that amount has, in my experience brought life into great peril; 2 cu. in. of arseniuretted hydrogen gas contains 1½ grains arsenicum. Arsenic is quickly eliminated in the excretions. I am of opinion that the presence of arsenic in the viscera is quite consistent with death from arsenic, the man having survived 2 days." To render occurrence of another such fatality impossible the firm took steps to substitute wooden utensils in the cleaning out of the stills for the metal ones previously used.

A few months later 10 cases of poisoning by AsH_3 were reported in the manufacture of zinc chloride. Zinc oxide—the waste from galvanizing pots—was treated with hydrochloric acid made on the premises in a vat situated in open air upon a platform. To protect the men from the weather a lean-to roof had recently been erected about 10 yd. away from the vat above a gangway along which the workmen wheeled the barrows of zinc oxide waste. This shelter played an unexpectedly important part in influencing the poisoning. On the occasion in question the men were eager to get through a certain amount of work by noon. The day was warm and unusually sultry. The fumes must have been drawn in the direction of the shelter by the indraught of a furnace situated in the building against which it was placed. The men working nearest to the vat itself suffered least of all; those in a well under the vat most; but seven men engaged in shovelling from a cart and never at any time within 10 yd. of the vat all suffered more or less severely, one dying on the seventh day after. The hydrochloric acid admittedly contained arsenic. Very decided information was obtained by Dr. Clayton of Accrington, who investigated the cases that others had previously occurred from time to time.

A similar case was reported in the manufacture of zinc sulphate in an uncovered vat in the open air which had only been used experimentally a few times. Subsequently the process was removed to a position whence all fumes could be conveyed to the main chimney stack. The necessity of carrying away gases likely to contain AsH_3 into the main chimney stack is emphasized by Dr. Dixon Mann after an investigation of five cases arising from the manu-

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facture of zinc chloride. The sulphuric acid contained 0.309 per cent. of metallic arsenic.

Galvanizing is a process where cases of AsH_3 poisoning might be expected in view of the quantities of commercial hydrochloric acid and zinc and iron used. Reported cases, however, are few. Seven cases with indefinite symptoms of headache, giddiness, sickness, weakness of the limbs and tremor having been notified, I visited the factory. The acute symptoms had passed off, but I satisfied myself that jaundice and hematuria had been present in some while others showed keratosis of the skin on the hands resembling the condition seen in poisoning from arsenic in beer. The only men who had been attacked were those engaged in dipping the iron sheets in the acid solution. The amount of arsenic in the acid used was stated to have been 0.035 per cent.—a rather small amount to account for the cases.

In the year 1912 an Inspector of Factories was summoned by telegram from the proprietor of a paper works who stated that a workman had been seized with symptoms resembling yellow fever. His work was to dissolve block tin after granulation in strong hydrochloric acid. After the pots were charged, steam was turned on to raise the mixture to boiling point. When solution was completed the pots stood for 12 hours to cool and the contents were then ladled out. During this last operation some gas was disengaged. The symptoms were typical—jaundice and hematuria. Samples of the acid, tin and zinc, and a sample of the urine also, 6 oz., were submitted to the Government Laboratory for analysis. The results as to the proportions of arsenic calculated as arsenious oxide were as follows:

Liquor from preparation bath	Practically nil.
Sheet zinc	Practically nil.
Block tin	0.03 per cent.
Feathered tin	0.05 per cent.
Precipitated tin	0.011 per cent.
Strong hydrochloric acid	0.20 per cent. or 159 grains per gallon.
Urine	0.07 grains per gallon.

The proportions of arsenic in the samples of block and feathered tin are those usually found in commercial tin, but the quantity in the hydrochloric acid is very high. The absence of arsenic in the preparation bath liquor (tin solution) indicates that the arsenic is given off in the form of gas or vapor during the process of solution of the tin in the acid, and direct experiments with the samples have shown that considerable quantities of arseniuretted hydrogen are evolved in this way. It is probable that most of the arsenic is given off as arseniuretted hydrogen. Assuming the samples to represent the materials generally used, the total amount of arsenic (calculated as arsenious oxide) evolved during each operation in which 132 gal. of acid are used is 3 lb., equivalent to upward of 10 cu. ft. of arseniuretted hydrogen.

The amount of the arsenic (0.07 grains per gallon) found in the sample of

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urine is much higher than that normally present in men employed in alkali works who may be supposed to run the chance of slight absorption and in whom the amount has been found to vary from 0.0011 to 0.0035 grain per gallon. At the works in question there were 700 earthenware vessels used in the room where the tin foil was extracted and fan ventilation was installed at the opposite end of the room to that where there are two open doors. Question of discontinuing the process altogether was under consideration. Use of de-arsenicated acid would remove the possibility of such accidents and should be pressed. Three cases occurred in a process for the recovery of copper, the men attacked being employed at the time in emptying vitriol from the depositing tanks. A fatal case was that of a man employed in a poorly ventilated room in dipping "store plates" composed of zinc with a small proportion of copper into a "bronzing" solution—essentially a solution of the chlorides of arsenic and iron dissolved in strong hydrochloric acid—to give the appearance known as "art metal."

Treatment.—The only form of treatment holding out any hope of success for the inhalation of arseniuretted hydrogen gas is prolonged inhalation of oxygen. Where there are facilities in a hospital, transfusion of blood from the artery of the giver into the vein of the sufferer and saline injections have been advocated.

Arsenical Poisoning from Consumption of Beer and Other Articles.—Brief note should be made of the circumstances attending outbreaks of arsenical poisoning from consumption of beer and other articles. On this subject the Report of the Royal Commission in England and Wales in 1901, following on the epidemic mainly in Manchester, Salford and Liverpool which affected certainly 6000 persons of whom at least 70 died, has given valuable data. The disease was characterized by paralysis and wasting of certain muscles, principally those of the extremities, and derangement of function in certain sensory nerves, closely resembling in these respects the peripheral neuritis associated with alcoholic poisoning. In addition, a great proportion of the cases exhibited various affections of the skin, such as erythemata, herpes, pigmentation, bullæ. In some the alcoholic type predominated and in others the arsenical. Beer contaminated with arsenic was accountable for all the symptoms. Analysis of some samples yielded as much as 1½ grains per gallon but usually the proportion found was from 1 to ½ to ¼ of a grain per gallon or less. The introduction of arsenic into the beer was made in the form of "brewing sugars"—glucose and "invert sugar"—which are extensively used as adjuncts to, or partial substitutes of, malt, and as "priming" solutions added after the fermentation of the wort. But both the glucose and the invert sugar derived their arsenical contamination from the acid used in preparing the former from starch and the latter from cane sugar. In the acid, responsible for the majority of the cases, as much as 1.45 to 1.9 per cent. of arsenious acid was detected. Sulphuric acid made from brimstone, or in the usual way after a de-arsenicating process

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effectively applied, may be practically arsenic free. This is not the only way, however, in which arsenic may contaminate beer. It may be introduced with the malt as the result of the arsenic in the coal or coke burnt in the drying of the malt. When the fuel is burnt the arsenic is volatilized and becomes deposited as arsenious acid on the surfaces of the kiln. Part remains in the ash and this also, if carried up by a draught of air, may settle on the malt. As bearing on this, six remarkable cases of arsenical poisoning caused by fumes from a coke stove in an ironing room of a laundry have interest. Vomiting and loss of power in the legs—in one case almost complete paralysis of them with complete paræsthesia below the knees, and loss of patellar and plantar reflexes—were the prominent signs without any changes in the skin. Two leaky coke stoves were used to heat the irons and while no carbonic oxide could be detected in samples of the air of the room there was a noticeable proportion of sulphur dioxide. The analyst suggested that as sulphur dioxide was present in the coke used arsenic was present also. He found 1/14 grain per pound (0.001 per cent.). Arsenic was detected in the urine of the two worst cases. Since coal only has been used in the stoves and leakage thus readily detected, there has been no more trouble.

The Royal Commission recommended that no substance should be used as an ingredient of food which contains a larger proportion of arsenic than 1/100 grain per pound, or, in the use of solutions, 1/100 grain per gallon.

The amounts of arsenic likely to be found in the urine during life or in the viscera post-mortem in cases of suspected industrial poisoning by arsenic are so small that, for its detection, recourse must be had to comparison of mirrors obtained preferably by the Marsh-Berzelius method after the material to be examined has been subjected to appropriate treatment by which any arsenic present is obtained in a solution suitable for the proper application of the test. By its means the Royal Commission state in their report that arsenic will be detected in amounts well below 1/1000 grain per pound or (in the case of a liquid) well below 1/300 grain per gallon. For an account of this method as also of the other recognized tests—Reinsch, Marsh and Gutzeit—reference must necessarily be made to standard works on toxicology.

An interesting point elucidated by the Royal Commission, and one of great value in obtaining indication of the past history of a patient in regard to arsenic, was the fact that persons taking comparatively small quantities of arsenic habitually excrete the poison in their hair to an extent readily appreciable by chemical tests. Thus men taking 3 minims of liquor arsenicalis three times a day (about 1/10 grain of arsenic daily) at the end of 2 months showed amounts of arsenic varying from 1/20 grain to 1/5 grain per pound in hair which had grown during the interval, whereas out of a total of 41 control cases the hair of 38 was either free from arsenic or showed no more than 1/150 grain of arsenic per pound of hair.

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SECTION II

BRASS, COPPER(?), AND ZINC POISONING

BY E. R. HAYHURST, M. D., Columbus, Ohio.

Copper and zinc, at least in toxic forms, are so infinitely bound up with the brass industry that it is well to consider them in this relation. The founding and casting industry in general is divided into two great divisions—iron and brass. Although the processes, except the manner of melting metals, are quite identical, still managements and tradesmen remain quite distinct. The brass worker, however, has an acquaintance with a great many more metals and alloys than the iron worker.

While nearly all metals may enter the brass crucible, copper and zinc are the primary ones and, with tin, constitute the ternary metals.

COPPER

Copper is mined extensively in various parts of the world, particularly the United States, China, Japan, the Ural Mountains and Sweden. It is practically the only metal which occurs pure,[1] as in the Lake Superior region. Usually it is in the form of various ores. The copper mines of the United States produce half of the world's product and employ over 53,000 men. The richest centers are Montana and Arizona, but Michigan mines employ the most men.[2] California and Utah are also important centers. Copper is worth in normal times about 15 cts. per pound. Copper melts at 1080°C., which is 350°C. higher than the point where zinc becomes a gas. Copper assumes a gaseous form at something over 1300°C.

Literature is replete with references to the relative harmlessness of copper upon the human organism. Certain vegetable organisms are extremely sensitive to the poisonous action of copper and its salts. Experimentally, it causes chronic poisoning when given in large amounts to animals.[3] It certainly has little significance as an industrial poison. "Copperman's chest"[4] is a pulmonary fibrosis, due purely to mechanical irritation. "Copper colic" and the rare cases of polyneuritis, affecting especially the lower limbs, reported[5] as occurring among brass polishers and coppersmiths and even those presumably working with pure copper, are rare complaints considering the large numbers so employed, and are probably due to lead,[6] arsenic, heat-fatigue toxins[7] or other factors.

Copper dust, or alloys rich in copper, generally produce in workers green-stained hair, greenish deposits on the teeth and in the gums, and a green tint to the perspiration, which may persist even after a thorough bath, while the

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skin may be actually bronzed. With the exception of sore gums and, perhaps, pyorrhea (these are both rare), the effects of all these upon the health are remote. Copper has been found accumulated[8] in all of the organs of the body, in the various excretions, and in the bones of former copper workers, which have shown a green color when subsequently removed from graves. Dogs fed copper pass it on to the suckling young,[9] in whose livers it may be found, without apparent effect. While not a normal constituent of the human body, copper is found in the liver in practically all autopsies because of the manifold opportunities of the human being to ingest copper.[10]

Workers in various copper processes are often thought to be "copper poisoned." While copper miners have various afflictions, none can be charged to any peculiar toxicity of copper itself. Zadek's[11] tables show that coppersmiths suffer above the average in most forms of sickness, but he discountenances copper poisoning. Seventy-five coppersmiths interviewed in Chicago[12] were uniformly healthy men, some of whom had been at the trade as long as 40 years. There was no evidence of "copper poisoning" among bronze or copper founders.

In copper smelting, sulphuric and other fumes must be acknowledged as obnoxious and very destructive to surrounding vegetation. In order to obviate this, very high chimneys are resorted to (one at Boston is 500 ft. high). The toxic vapors which escape from the electrolytic refining of copper (an important American industry) are acidic in character, and cannot be considered as copper poisoning. While copper volatilizes to a slight extent below its boiling point (probably the surface temperature of the molten mass actually reaches above the boiling point), the metal is rarely boiled, as is zinc, in any industrial process. In an instance in which this did occur (electric furnace),[13] those who inhaled the vapors suffered from severe influenzal-like symptoms, quite similar to those experienced after inhaling zinc fumes. Perhaps the severe symptoms experienced by those who breathe the fumes arising from pots of German silver (copper 60, zinc 24, nickel 16) are due to some extent to volatilized copper, as well as to zinc, since the melting point of nickel (1500°C.) is above the boiling point of copper. Certain salts of copper are astringent, or even caustic, and, in chemical industries and copper plating, may produce contact poisoning. The idea that copper workers are immune to typhoid fever was exploded 30 years ago.

ZINC

Zinc occurs in nature principally as the carbonate (calamine), sulphide (blende), and silicate. Its ores are widely distributed.[1] It was first smelted in England in 1743, in Europe in 1807, and in the United States in 1873. The United States led all other countries in 1911 with nearly 3,000,000 tons production. The American industry engages over 21,000 men (1910),[2] or 2 per cent. of all miners, and is increasing at the rate of 20 per cent.

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annually. Smelting is usually done in the nearest coal districts. The Western ores contain[3] lead, iron, cadmium, and occasionally arsenic. The New Jersey ores are free from arsenic, practically so from lead, but contain manganese.[4] The price of zinc, or "spelter," keeps at about 5 cts. per pound, and is controlled by an international syndicate.[5] Since the onset of the European war it has been quoted as high as 37½ cts. Zinc melts at 417.6°C. and boils at 730°C.; hence it is, with the exception of cadmium, the only supposedly innocent metal boiling under 1000°C., an important point to remember since metal workers are thus industrially liable to the volatilization products of but one metal—zinc. In fact zinc volatilizes at temperatures as low as 500°C.[6] similar to the well-known evaporation of water at temperatures far short of its boiling point. The use of chlorides (as in galvanizing) still further promotes the volatilization of zinc. It is important to remember that all metals distill to a limited degree far below their boiling points, and if the metal is intrinsically poisonous its mere molten condition becomes a health hazard, which increases with temperature. Lead, for instance, distills even more readily than zinc, although its boiling point is 900°C. higher.

Zinc is the most wasted of all metals.[7] It is also the most impure of all metals used industrially and, on this account, retains its old name "spelter."[8] There is usually present a trace of lead (less than 0.1 per cent.) as well as iron, and sometimes manganese, cadmium and arsenic. But these impurities are very rarely the cause of industrial poisoning. Plumbism in this relation is usually due to the fact that lead is subsequently added to spelter alloys.

Tests for Zinc.—Qualitatively Ganassini's[9] test is very delicate. The substance to be tested, preferably in the form of zinc sulphide or calcium zincate, is exposed for a moment to the fumes of boiling hydrochloric acid arising from a test tube. The substance is then heated briefly to drive off chlorine fumes. A uric acid reagent is made by dissolving 2 grams of uric acid in 100 cc. of 5 per cent. KOH. Streak the substance with this reagent, using a fine glass filament. Then touch the moistened streak with a crystal of K_3FeCn_6, or with a freshly prepared 10 per cent. solution of the same. In a moment a beautiful azure-green color will appear. Avoid excess of the uric acid reagent. In verifying test reagents try them directly upon a piece of zinc or galvanized iron. Slight modifications render the test still more delicate and micro-chemical in character. The test may be applied directly to washed mucous membranes and sections of organs. The most delicate quantitative test[10] is the calcium zincate method which, however, is a little too extended for the general practitioner. Its delicacy is claimed to be one in 30,000,000 parts, even in the presence of other metals and salts.

Principal Uses of Zinc.—Zinc has the following principal uses from the sanitarian's viewpoint: (1) Processes involving the use of brass and bronze alloys; (2) galvanizing; (3) manufacture of German silver and white metal; and (4) the use of zinc oxide (zinc white), used especially in the rubber and paint industries and the handling of zinc salts. Other uses of zinc are as

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lithopone, a lead substitute in paints and enamels; in the manufacture of oil-cloth; the manufacture of galvanic battery plates; the de-silverization of lead; etching and lithographing plates; and the manufacture of parchment paper.

Toxicity of Zinc.—Zinc per se is non-poisonous.[11] In this, practically all recent authorities are agreed. Michaelis[12] claims zinc to be a normal constituent of human tissues (probably in very minute quantities). Zinc exists in a large number of plants in which it probably has a definite physiological function.[13] In animal tissues, zinc causes precipitation in unusual dilution,[14] forming an inert(?) albuminate. Zinc ions markedly activate metabolic processes.[15] When taken internally, zinc as oxide or salt can be detected in the blood and practically all tissues of the body.[16] Given subcutaneously, the same obtains. Zinc does not accumulate to any extent in the system, but it is stored temporarily in the liver and is found in the bile, urine, milk and other excretions. As with other metals, it is more largely excreted by the intestines, and perhaps the stomach, than by the kidneys.[17] Zinc albuminate does not affect red corpuscles nor other tissues, except possibly the heart muscle (frogs).[18]

Certain salts of zinc, as the sulphate and chloride, are well-known astringents and caustics, but industrial poisoning (dermatitis, conjunctivitis) occurs from these only as the result of carelessness in their handling. In their manufacture arsenic poisoning may occur from impurities. Zinc chloride is used for denaturing alcohol, as a preservant for wood, for preparing parchment paper, and for weighting goods. The fumes of zinc chloride which form in soldering zinc, previously cleansed with hydrochloric acid, are an occasional complaint (proteid resorption poisoning ?). Zinc sulphate is used in varnishes and as a mordant for the production of colors on calico. It is also applied with a brush, in saturated solution, to plaster and cement surfaces as a preparation for subsequent painting.

But there is something which occurs in association with the immediate products of zinc fumes which causes acute intoxication. Molten zinc when slightly overheated produces a greenish-yellow flame, and yields to the air a whitish smoke, composed of the flaky oxide of zinc ("philosopher's wool"). This is usually encountered in the making and pouring of brass. There is also produced some zinc dust, due to incomplete oxidation. Obviously the fumes themselves, being at high temperature, cannot be inhaled or at least absorbed as such. Experimentally, zinc dust, zinc oxide and zinc carbonate are all harmless,[19] whether ingested or inhaled. Lehmann,[20] however, proved that the symptom-complex known as "brassfounders' ague" could be produced in every feature in man by burning pure zinc and inhaling the oxide therefrom. He believes that the zinc oxide produces proteid destruction in the respiratory membranes with resorption of the destroyed cell contents into the system, and that these dead proteids acting as toxalbumens produce the symptoms. Experimental animals do not develop the

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same symptoms, but after such exposure show a more or less violent congestion and reddening of the respiratory mucous membranes with zinc demonstrable in the lung tissues and, later, in various organs (guinea-pigs and rabbits).[21] To our mind, the phenomenon is best explained as due to the hygroscopic action of the oxide (or zinc carbonyl, $ZnCO_4$)[22] in the hot foundry atmosphere. Thus the usually inert oxide not only dries but actually sears the epithelium (ciliated, simple and parenchymatous) of the respiratory mucous membranes. An analogy is seen in the fact that zinc chloride may even carbonize organic substances in its demand for moisture, and the resultant symptoms (as seen in intra-uterine cauterizations[23] with zinc chloride) are similar to, and also explainable as, proteid resorption phenomena. There are many things in the composite picture of "zinc ague" which tend to bear out the above explanation: (1) Pharmacologists have proven the harmlessness of non-caustic zinc compounds;[24] (2) the quick immunity to subsequent attacks, which is similar to all foreign-proteid poisonings; (3) no poisonous metal is known to produce a rise in temperature; (4) metallic poisons act accumulatively; (5) infection is negatived since ulcerations, septicemia, pyemia, and fatalities never occur; (6) fumes of other metals, whether such metals are poisonous or not per se, produce similar symptoms (copper,[25] nickel,[26] iron,[27] cobalt,[28] cadmium[29]); (7) that the symptoms are associated with zinc (or brass) only is simply a trade-process circumstance, due to the low boiling point, excessive volatility, cheapness and therefore waste of zinc to the air.

ZINC AGUE

Synonyms.—Brassfounders' ague, brass chills, zinc chills, smelter shakes, metal shakes, zinc asthma, braziers' disease.

Definition.—"Zinc ague," or "brassfounders' ague," may be defined as an acute malaria-like syndrome of chill, fever (sometimes) and sweat, appearing a few hours after the inhalation, for a few minutes or longer, of zinc fumes, whether pure or, as is the usual rule, in the form of brass fumes, affecting only, or mostly, those unaccustomed to such exposure; further characterized by the development of a form of temporary immunity, and absence of immediate serious or fatal consequence. Definite chronic symptoms due to the presence of zinc probably do not occur, but the morbidity and mortality rates of workmen constantly exposed to the breathing of the fumes are high, with respiratory diseases especially in evidence.

Occurrence.—Of the different classes of workers, the symptoms of the "ague" do not occur among the miners, nor in the handling of zinc or its compounds at ordinary temperatures; they may occur, though rarely, among galvanizers and infrequently among zinc smelterers and oxide makers; they occur very commonly among workers in brass foundries, casting shops and in brazing processes, also among refiners of junk metal and dross.

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Symptoms.—At the end of a few hours' exposure, or even less, to the breathing of the metallic smoke which arises during the melting or pouring of brass or zinc, the nose, throat and substernal region have a sensation of dryness, soreness and burning. The conjunctivæ feel irritated. There may be a metallic taste in the mouth, an unproductive cough, a feeling of constriction in the chest, lassitude, headache and loss of appetite, followed sometimes by nausea and vomiting. Within one to several hours after the exposure the vision may become blurred, the headache worse, the cough more productive and the sputum blood-streaked.[30] Chilly sensations begin to appear and the victim usually takes to his bed. The shivering rapidly increases into a distinct rigor, which may last from ½ to 2 or 3 hours. No amount of external heat or friction appears to lessen the chill and shaking. There is a great desire to imbibe hot drinks and stimulants. During this stage there are usually muscular cramps and sharp joint pains. The victim feels that he is deathly sick. These symptoms end almost by crisis and are followed by a most profuse perspiration, in which some claim there is an odor of zinc. There is an immediate sensation of great relief. Sleep supervenes and the victim awakens the next morning with no remaining symptoms of his recent experience other than a little exhaustion, perhaps a metallic taste, and a temporary loathing of food. The entire attack lasts from a few to 20 hours, seldom longer.

In many cases there is a distinct rise in temperature during and following the chill, but it is not constant and may be subnormal. It does not usually mount high. The pulse during the chill is small and rapid—120 to 130 per minute (zinc effect upon the heart ?). The respiratory rate is not much affected. The blood findings during and after a chill have been reported as normal, although some have found a slight leucocytosis, and Arnstein,[31] in experiments with brass fumes on men, found a constant leucocytosis to 16,000, in which neither the per cent. of lymphocytes nor of eosinophiles was affected, thus differing from an infection. Sigel[32] proved the absence of carbon monoxide hemoglobin as an etiological factor by spectroscopic examination of the blood. The urine after a severe chill invariably shows albumen and casts which clear up within a day or so, and zinc is usually present, although much more of this metal is to be found in the feces. Arnstein was struck by the fact that these symptoms were evoked by very minute traces of zinc in the air (0.007 gram of zinc oxide in 30 liters of air). Graeve[33] reports an enlargement of the spleen noticeable after a few days.

There are many conditions which seem to influence the onset and severity of zinc chills. The newer workmen, or those who have returned to work after a week's vacation, perhaps only after a day or so off, as over Sunday, are the most liable. Unless the exposure to the metallic smoke and vapors has been unduly severe, the workman continues at his vocation all day without noticing untoward health effects. It is usually after leaving work at night that the sickness comes on. Many state that as soon as the cold out-

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side air strikes them or when removing the clothes at bedtime the chill is inaugurated. Hence chills are far more common in winter than in summer. This seasonable influence is due mostly, however, to decreased ventilation through closing of doors, windows and skylights in foundries. Following a snowstorm, such a large per cent. of workers have been seized with chills that the condition has been mistaken for an epidemic of some form. The worst complaints in my experience have come from so-called bronze foundries where journals are cast for car wheels. Although the bronze contains only a small per cent. of zinc, it is melted and poured by the ton so that the amount of fumes becomes excessive. Lead poisoning from babbitting is very frequent here too. The chills occur rarely in shops where pouring is done but once or twice daily. The frequency of the chill, other conditions remaining the same, is almost in direct ratio to the percentage of zinc contained in the alloy. Furnace men, casters and helpers are rather more exposed than moulders. Deep breathing while under strain is a most important factor. About 70 to 80 per cent. only of workmen seem susceptible; hence some natural immunity seems to exist. Also a tolerance occurs in about 70 to 75 per cent. of workmen who work steadily at the trade. A further 20 to 25 per cent. sicken more or less regularly, but usually not severely. Only in very rare cases do these attacks occur so frequently that a change of occupation is necessary. Still many older workmen prefer to lay off in the winter season. The workers consider the chills so commonplace and matter-of-course that they do not go or send to the physician on account of them. All factors which tend to lower natural vitality are predisposing to zinc chills, especially alcoholism,[34] poor quality of food, as is consumed by a large proportion of the foreign workmen, irregular hours, and excesses of all kinds. In addition, weak, anæmic individuals, young persons and women are more subject to zinc fumes than are robust individuals.

Unlike lead, zinc does not produce chronic poisoning. This is best explained by the fact that it has very little tendency to accumulate in the system. The chronic neuritic symptoms, described by older writers as affecting the lower extremities particularly, were quite probably due to impurities in the metals, such as arsenic and lead. However, the continual occurrence of zinc chills produces, in from 5 to 20 years' time, the same results as described below in brass poisoning. (The morbidity and mortality statistics of zinc workers are considered under the special industries in Part II.)

Diagnosis.—As just stated, the physician must get away from the idea of attempting to diagnose chronic zinc or brass poisoning, as there is no such condition. Look to lead, arsenic or manganese for the specific causes of such conditions. Every complaint of chill, fever and sweat, lasting a few hours' time, and with absence of interval symptoms should cause inquiry to be made as to exposure to metal fumes, the more so if the symptoms occur after the work period, or during inclement weather, or the winter season.

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The worker himself may consider the symptoms due to malaria, especially if he has ever been so afflicted. Zinc ague attacks must be differentiated from influenza, or la grippe, as there is respiratory complaint, aches and pains, and a subfebrile movement in all three, but the "ague" shows a minimal character of catarrhal symptoms with pain, soreness and constriction symptoms in the throat and substernum prominent. The chills are usually unduly severe, while the recovery from subsequent malaise after the sweating period is quick, and there is an entire absence of sequellæ. Acute bronchitis, onset of tonsillitis, or of septic processes may confuse for the first few hours. The repetition of the chills and fever of irregular type may be confused with the course of tuberculosis, Hodgkin's disease and leukemia, while, on the other hand, the physician often comes to the erroneous conclusion, particularly if no fever can be detected, that he is dealing with a case of neurasthenia, hysteria, or malingering.

BRASS

Brass is an alloy of copper with a base metal, usually zinc, but sometimes tin, and contains varying quantities of other metals. Fine brass or red brass consists of copper 2 and zinc 1. However, as these two metals combine in practically all proportions, many alloys are possible. "Cheap yellow brass" is very rich in zinc. The making of brass was known to the Phœnicians before Solomon's time. Pliny describes brass fumes. The brass of the Bible was probably bronze. From ½ to 13 per cent. of lead is contained in brass alloys.

Bronze is an alloy consisting of copper 9 and tin 1. In the industry, however, red brass is very often styled "bronze," although zinc is the other component instead of tin. Many metals and metalloids are added to brass and bronze to convey certain physical properties. Aluminum, copper and German silver castings are other products.[1]

Usually scrap metal consisting of all sorts and compositions of (nonferrous) metals makes up one-third or more of the alloy; then copper, zinc, or lead ingots are added, to arrive at an approximate composition. Consequently it will be seen that the brass manufacturing industry, or more properly the non-ferruginous metal-working trade, is engaged in the handling of large amounts of copper, zinc and tin, while it must not be overlooked that lead, antimony, nickel, aluminum, cadmium, manganese, iron, phosphorus, arsenic and sulphur may enter the brass crucible. In subsequent processes of cleansing, plating and finishing, dangerous acids and cyanide solutions are used.

Pseudo-brass Poisoning—Wounds due to brass objects and similar alloys are of traumatic and infectious significance only. Brass dust (grinders, polishers and buffers) does not produce "brassfounders' ague," and has only the harmful effects of metallic dusts in general. The lead in the alloy-dust may produce its symptoms in very susceptible persons(?). Brass in any con-

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dition short of the recent vaporous state of its components produces no intoxication peculiar to itself.

The essential health-hazard from brass is the inhalation of the metallic fumes. Analyses of brass fumes show:

Substance	Deposits from flues[2] A	Deposits from flues[2] B	Analysis of fumes[3] C	Bench-settling[4] D
Zinc oxide	32.13	24.74	28.82	(zinc) 44.9
Lead oxide	0.31	1.92	0.00	(lead) 0.8
Iron oxide	2.43	....	2.78	....
Copper oxide	2.85	2.50	(copper) 1.71	....
Cadmium oxide	1.56	0.89	....	....
			(moisture) 9.64	....

Insignificant traces of arsenic, nickel and manganese may be present, and Sigel found the chills where copper was absent in the fumes. Symptoms traceable to phosphorus in phosphor-bronze and manganese in manganese-bronze have never been reported. Antimony poisoning occurs, however, among type-metal refiners and melters, and dermatitis and "biting" naso-tracheitis from this should be distinguished from "brass itch" and "zinc asthma" respectively. Braziers and hard-solder workers, especially where electric welding or the modern blow pipes (oxy-acetylene, oxy-hydrogen) are used, may suffer greatly from metal chills due to volatilization of the alloys worked upon.[5]

Brass Poisoning.—"Brassfounders' ague" is the only condition which can be recognized under this heading and this has been described under zinc where it properly belongs. The condition was first mentioned by Thackrah in 1832,[6] who considered it periodic because seasonal; the zinc factor was recognized by Greenhow[7] in 1862; the earliest American cases were cited by Oppenheim[8] in 1894, while later references are Moyer and Lavin,[9] also Pietrowiecz,[10] Chicago, 1904, Sicard,[11] New York, 1905, and Hayhurst,[12] Illinois, 1910 and 1912.

Chronic Effects of Brass Poisoning.—In Chicago[13] the fact that 85 per cent. of 1761 foundry workers were under 40 years of age, and only 1 per cent. over 50 years, was explained by employers as due to "slowing up," or beginning increpitude, and by workmen, as gradual incapacitation from the inhalation of brass fumes and the strain of the work. The constant intake and elimination of zinc from the system along with the repetition of brass chills or the constantly forced immunity to the same are enough to cause degenerative diseases in themselves even though the immediate afflictions above mentioned are overlooked. Chronic bronchitis or "asthma," emphysema and pulmonary tuberculosis are very common. Older workers invariably complain of dyspepsia, "biliousness," occasionally of gall-stones

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and they are often icteroid. Constipation is the rule among them and hemorrhoids are frequent complaints. Pyorrhea alveolaris, carious teeth, sallow complexion and anæmia, ill-nourishment and emaciation, chronic alcoholism, Bright's disease, nervous and heart diseases are all above the average.[14]

Remedial Measures.—No specific treatment for zinc or brass ague has been discovered. Mild emetics, hot milk, and stimulants are used. A good purge seems as beneficial as anything. To counteract the effects of the sublimed fumes which are swallowed the drinking of milk before and after pouring, and of water containing sodium bicarbonate, magnesia or lime water, is recommended. For such as is inhaled, particularly if a chill is anticipated, a dose of Epsom or Glauber salts should be taken at once, and to relieve the respiratory membranes an expectorant, such as ammonium chloride, combined, perhaps, with Brown's mixture. During the course of the chill, hot lemonade, hot milk, hot milk with pepper, seem welcomed. The use of whiskey, or any alcoholic liquors could be of value only because of their narcotic action, and according to the statements of the more observing workmen are of no more benefit nor relief than the hot drinks above mentioned. Further, alcohol increases the subsequent malaise, and adds to the liability of sequellæ. Alcoholic beverages are especially to be discouraged because of the development of industrial alcoholism, that is, the worker who has come to believe that alcohol is a true antidote to the metal fumes, rapidly becomes a chronic alcoholic as a result. Many such cases are seen. This condition only lays him more liable than ever to the "ague." In fact many workmen claim that abstainers are the last to succumb to the chills, and have them the mildest. The prostration usually compels one to take to bed, where plenty of covering and external heat, especially to the feet, are very comforting. Antipyretics and antiperiodics are useless, while the symptoms do not usually warrant opiates. Finally, any physician may consider himself lucky who has been called to see a case of "zinc ague" at its height, especially in an informed worker, since the victim usually knows what he has got, and also its cause, and immediate prognosis, and hence does not see the need of the services of a physician. But the physician will see many cases of tuberculosis, pneumonia, chronic degenerative diseases and pre-senile conditions if in practice much among zinc or brass workers who are concerned with the hot processes. (The prophylaxis of "zinc ague" is considered in Part II.)

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BRASS

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THE HEALTH OF BRASS WORKERS

BY T. M. LEGGE, M. D., London, England.

In the year 1902 detailed inquiry into the health of brass workers in Birmingham—the center of the industry—was undertaken by the Factory Department of the Home Office because not only was there ignorance of the real prevalence of so-called brassfounder's ague among them, but also because the view had been expressed that a definite clinical picture was associated with filing, polishing or otherwise manipulating brass, of which the prominent symptoms were a green line on the teeth, anæmia and emaciation.

In order to obtain accurate information as to illness attributable to poisoning by brass I undertook the examination of 233 casters (among whom were included metal mixers, casters proper, and casters' helpers), and 210 polishers and others (including polishers proper, dressers, turners and burnishers). The object throughout was to make comparison between those exposed to fumes and those not so exposed. In addition to the filling in by each one of the men of a printed form as to the state of his health while working in brass note was made of the height, weight, chest measurement and strength of grasp as tested by a dynamometer; the heart, lungs, gums and teeth, were examined; and the condition as to anæmia, paresis, general health, and prevalence of brassfounder's ague, was ascertained. The inquiry was carried out in 21 factories in Birmingham in all of which, with one exception, both zinc and copper entered largely into the metal poured. Subsequently I visited the principal foundry in the Royal Arsenal, Woolwich, and examined 89 persons of whom 58 were engaged in casting gun metal into the composition of which zinc hardly enters at all.

Tabulation of the results as regards age, duration of employment, height, weight and chest measurement, showed differences so slight that generalization from them revealed nothing of importance. The strongest evidence perhaps obtained in the inquiry that the operation of casting is more trying than other brass work came out from the replies to the question, Do you consider that your health has been in any way injured by working in brass? since 22.7 per cent. of the casters as compared with only 11.6 per cent. of the polishers said they had suffered. Of 193 casters interrogated as to brassfounders' ague 123 or 63.7 per cent. stated that they had suffered more or less frequently from the train of symptoms—malaise, sense of weakness, shivering with chattering of the teeth and extreme feeling of cold—characteristic of this complaint. There could be no doubt as to the extreme prevalence of the affection. The frequency of the attacks in several cases—often once

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a week—was remarkable, the attack, as has so frequently been described, following on return to work after temporary absence for a day or two.

There was general censensus of opinion that the greater the proportion of spelter in the metal mixture the greater was the liability to attack. The strongest evidence I obtained on this point was at Woolwich Arsenal. Among the 58 casters of gun metal where conditions of ventilation in the past would have led one, if anywhere, to expect heavy incidence of brassfounders' ague six only narrated indefinite symptoms suggesting the possibility that they had suffered. In another foundry at the Arsenal, however, where zinc entered largely into the composition of the metal, of the 10 casters questioned all but one gave a history of frequent attacks.

It was impossible not to be struck by the less satisfactory appearance of the casters as compared with the polishers. By the sallowness of the complexion the occupation as a caster could often be correctly guessed.

Evidence of lead absorption—showing itself in a blue line on the gums—was noted in four casters and in 16 polishers. In one man engaged in polishing on an emery wheel unprovided with exhaust ventilation paralysis of the wrists was found. Analyses of two samples of brass dust made in the Government Laboratory showed in the one case 2.9 per cent. of lead and in the other 2.43. I have no doubt that inhalation of brass dust containing so small a percentage of lead as these samples show can in the long run give rise to plumbism.

A green line on the teeth from formation of basic carbonate of copper was noted in at least 65 per cent. of the polishers and 16 per cent. of the casters. It is, therefore, more an indication of work involving exposure to dust rather than to fumes. Greenish coloration of the hair was noted twice.

The conclusion at which I arrived from the inquiry was that work in the casting shop was more trying and attended with greater injury to health than work in polishing. Apart from brassfounders' ague and the occasional development of lead poisoning among chandelier fitters and polishers, I did not find symptoms of illness which suggested to my mind that they could possibly be due to absorption of copper or brass. If, therefore, so-called brass poisoning were made notifiable in the same way as poisoning by lead, arsenic, mercury and phosphorus, the result would be the reporting of any ill-defined sickness in persons showing a green line upon the teeth or even of persons working merely in brass. Mortality statistics, however, as for instance those contained in the Decennial Supplement of the Registrar General, show that there is undue incidence of phthisis among those employed in brass working. What was wanted in casting shops, I said, was removal of the fumes and in polishing shops removal of the dust. At the time I investigated the subject there were not more than three or four out of the 900 casting shops in Birmingham in which attempt was made to remove the fumes locally by exhaust ventilation and in the polishing shops only one.

Subsequently in 1907 the Home Office issued Draft Regulations in

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accordance with the requirements of the Factory and Workshop Act, 1901, which it was proposed should apply to factories in which the process of casting was carried on. Numerous objections from manufacturers were received (in consequence perhaps of ambiguous wording in the Draft) and a Commissioner was appointed by the Secretary of State to hold a public inquiry in regard to the same under section 81 of the Act. At the inquiry strong evidence from casters as to the effect of the fumes on themselves was heard, but perhaps the most striking evidence was that of the Commissioner's own experience as stated in his report:

> On the occasion of one of my visits to Birmingham, after several visits to casting shops and seeing some pourings, I inspected toward the close of the day a strip casting shop, where the ventilation proved to be extremely bad. I witnessed four crucibles of brass, containing 120 lb. each, poured in succession into strip moulds, the doors and windows being meanwhile closed for reasons hereinafter referred to. The time occupied was some 15 minutes. On coming out into the air I felt a sense of stifling oppression on the chest, which passed away in about an hour, a strong taste of the fumes, however, remaining. At about 10.30 P.M. the same night symptoms corresponding exactly with those of the "brassfounders' ague" or "fever" showed themselves: shivering and chattering of the teeth followed by what appeared to be a considerable degree of fever, which lasted for perhaps 2 hours, then a most profuse and long-continued perspiration, followed at last by sleep. In the morning, however, I rose at the usual hour with only a slight headache and a taste of the fumes in my mouth. Mr. Thornton Lawes, who had been with me throughout the previous day on my tour of inspection, and had witnessed the casting to which I have referred, had a similar attack of the "ague" or "fever" the same night. On that or the next day I began to feel pains in the shoulders and chest, and for over 2 months I have suffered from these and from asthmatic and bronchial symptoms which have been sufficiently severe to compel me to give up my work and seek a cure in the country.

As the outcome of this Inquiry the Regulations which are appended to this article were issued by the Secretary of State in 1908 and now govern the industry. In Birmingham alone they apply approximately to 860 casting shops of which about 320 are exempted by reasons of the exception with regard to air space per person. Most of the casting shops are ground floor rooms with lean-to roofs made of slate. Many small and unsuitable shops have been discontinued during the past few years. The most popular method by which compliance with the Regulations as to exhaust ventilation has been secured is provision of a large hood fixed about 6 or 7 ft. above the floor connected up with a power-driven volume fan. The moulds are placed in a leaning position against a shelf or "spilling hearth" 10 in. or more above the floor directly under the hood and, whilst pouring, the pot is rolled along this shelf behind the moulds. Another common device in use—the most successful form of locally applied exhaust ventilation by heat which I know—is that invented by Messers Rainsford and Lynes, Ltd., brass casters, of Birmingham. In this case a small cap is attached to the crucible containing

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the molten metal and connected by a flexible pipe some 3 or 4 in. in diameter to a fixed tube running into the furnace chimney. With this apparatus no fan is necessary and yet, in the act of pouring, at least 85 per cent. of the fumes never enter the workroom at all. Unfortunately its use is limited to sand casting as it is alleged that it cannot deal with the enormous volume of zinc oxide fume given off in strip or tube casting. The illustration shows the mode of application. Progress, however, in regard to exhaust ventilata-

FIG. 1.—New method of pouring in brass casting; 85 per cent. of the fumes are taken away.

tion has been so rapid in recent years that all manner of devices now enable occupiers to comply with the requirement as to removal of the fumes.

While the exemption from the Regulations allowed on the ground of ample cubic space per person was necessary, there cannot be question that, in comparatively small casting shops, work is carried on under healthier conditions where there is a fan to induce a constant current of fresh air through the room while pouring is going on than where, owing to the exception, reliance is placed on natural means of ventilation only.

Commencing in 1902 and from that year onward the inspectors of fac-

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tories in Birmingham and elsewhere had been pressing, in brass-polishing shops, for action in pursuance of section 74 of the Factory and Workshop Act, 1901, which states that if polishing on a wheel is carried on so that dust is generated and inhaled by the workers to an injurious extent, and it appears to an inspector that such inhalation could to a great extent be prevented by the use of a fan, the inspector may direct that a fan be provided within a reasonable time. A spirit of emulation among the employers was set up and without recourse to legal proceedings having been necessary, it is now safe to say that exhaust ventilation is applied at all benches where polishing is continuously done. Although from time to time I had when in Birmingham seen admirable instances of the success of the Regulations, it was not until the beginning of the year 1914 that I had opportunity of revisiting the factories in which I had made my original inquiry. I then found in every casting shop, not exempted by reason of air space, suitable provision made, in one or other of the ways described, for speedy removal of the fumes. Several of the casting shops were small and incidence of brassfounders' ague had been severe, but the same men whom I had examined under the old conditions were insistent as to the striking improvement in the conditions of work which had resulted. Brassfounders' ague in their experience had become, practically speaking, a matter of historical interest only. The amelioration in health, so I was informed, was reflected in the lessened amount of sickness claims made on their Society. The single instance of complaint brought to my notice was in a large casting shop where attempt had been made to arrange a hood and fan over spilling hearths in the center of the room. Here natural cross currents at times defeated the draught induced by the fan and explained the partial failure.

In conclusion, I would refer to the interesting experimental work published by Professor K. B. Lehmann of Würzburg which has, in my opinion, thrown much light on the causation of brassfounders' ague. By experiment on the human subject (himself, his assistant, and a brass caster peculiarly susceptible to the malady who volunteered) he was able to produce all the typical symptoms of brassfounders' ague by inhalation of the fumes of burning zinc alone in a badly ventilated room. His conclusions were that the malady could be very easily produced in men working with chemically pure zinc while it is burning. The symptoms observed resembled closely the effects of bacterial infection through the respiratory system. Lehmann believes that some of the cells lining the respiratory tract are destroyed by the zinc oxide fumes and it is absorption of these dead cells which accounts for the symptoms. The objection raised to the zinc theory, which at first sight seems difficult to disprove, is based on the fact that spelter workers do not suffer. The probable explanation of this is that while zinc melts at 400 to 500°C., brass requires a much higher temperature than this to melt. When producing brass, the copper is melted first at a temperature of about 1000°C. and the zinc is added afterward. The higher the temperature the

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more rapidly does the zinc burn and the stronger are the oxide of zinc fumes given off.

REGULATIONS, DATED JUNE 20, 1908, MADE BY THE SECRETARY OF STATE, GREAT BRITAIN, FOR THE CASTING OF BRASS

Whereas the casting of brass or any alloy of copper with zinc has been certified in pursuance of Section 79 of the Factory and Workshop Act to be dangerous, I hereby in pursuance of the powers conferred on me by that Act make the following Regulations and direct that they shall come into force on the 1st day of January, 1910, and shall apply to all factories in which the casting of brass is carried on, with the following exceptions:

(i) The Regulations shall not apply to a sand-casting shop having an air-space equivalent to 2500 cu. ft. for each of the persons employed nor to any other casting shop having an air space equivalent to 3500 cu. ft. for each of the persons employed. Provided—

(*a*) that provision is made for the egress of the fumes during casting by inlets below and outlets above of adequate size, and

(*b*) that a notice in the prescribed form, giving the prescribed particulars, shall be kept affixed at or near the entrance of the casting shop and that a copy thereof shall be sent to the Inspector of the district, and

(*c*) that the conditions of exemption stated in such notices are not departed from.

(ii) So much of Regulation 1 as requires that exhaust draught shall be maintained during the process of casting shall not apply in the case of strip or solid drawn tube casting or any other class of casting which the Secretary of State may certify on that behalf, provided that—

(*a*) the exhaust draught cannot be so maintained without damage to the metal (proof of which shall be upon the occupier); and

(*b*) the exhaust draught is put into operation immediately after the casting; and

(*c*) provision is made for the egress of fumes during casting by inlets below and outlets above of adequate size.

(iii) Where it is proved to the satisfaction of the Chief Inspector of Factories that by reason of exceptional features in the construction or situation of a casting shop or by reason of the infrequency of the casting or the small quantity or the nature or composition of the metal cast or other circumstances all or any of the Regulations are not necessary for the protection of the persons employed he may by certificate in writing (which he may in his discretion revoke) exempt such casting shop from all or any of the provisions of the same subject to such conditions as he may by such certificate prescribe.

In these Regulations (including the above provisions and exceptions)—

"Brass" means any alloy of copper and zinc.

"Casting" includes the pouring and skimming of brass.

"Casting shop" means any place in which casting of brass is carried on.

"Sand-casting" means casting in moulds prepared by hand in sand or loam or sand and loam.

"Sand-casting shop" means a place in which no kind of casting other than sand-casting is carried on.

"Pot" includes any crucible, ladle or other vessel in which the brass is skimmed or from which it is poured.

"Employed" means employed in the casting shop in any capacity.

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"Persons employed" means the maximum number of persons at any time employed.

It shall be the duty of the occupier to observe Part I of these Regulations, and the conditions contained in any certificate of exemption.

It shall be the duty of all persons employed to observe Part II of these Regulations.

Part I

Duties of Occupiers

1. Casting of brass shall not be carried on unless the following conditions are complied with:

   (*a*) There shall be an efficient exhaust draught operating by means either of (i) a tube attached to the pot, or (ii) a fixed or movable hood over the point where the casting takes place, or (iii) a fan in the upper part of the casting shop, or (iv) some other effectual contrivance for the prompt removal of the fumes from the casting shop and preventing their diffusion therein. The exhaust draught shall be applied as near to the point of origin of the fumes as is reasonably practicable, having regard to the requirements of the process, the maintenance of the exhaust draught during the process of casting, and (as regards casting shops in use prior to 1st January, 1908) the structure of the premises, and the cost of applying the exhaust draught in that manner.

   (*b*) There shall be efficient arrangements to prevent the fumes from entering any other room in the factory in which work is carried on.

   (*c*) There shall be free openings to the outside air so placed as not to interfere with the efficiency of the exhaust draught.

2. There shall be provided and maintained in a cleanly state and in good repair, for the use of all persons employed, a lavatory, under cover, (i) with a sufficient supply of clean towels, renewed daily, and of soap and nail brushes, and (ii) with either—

   (*a*) A trough with a smooth, impervious surface, fitted with a waste pipe without plug, and of such length as to allow at least 2 ft. for every five such persons, and having a constant supply of warm water from taps or jets above the trough at intervals of not more than 2 ft.; or

   (*b*) At least one lavatory basin for every five such persons, fitted with a waste pipe and plug or placed in a trough having a waste pipe, and having either a constant supply of hot and cold water, or warm water, laid on, or (if a constant supply of heated water be not reasonably practicable) a constant supply of cold water laid on and a supply of hot water always at hand when required for use by persons employed.

3. No female shall be allowed to work, in any process whatever, in any casting shop.

Part II

Duties of Persons Employed

4. No person employed shall leave the premises or partake of food without carefully washing the hands.

5. No persons employed shall carry on the pouring of brass without using apparatus provided in pursuance of Regulation 1 (*a*).

6. No person employed shall in any way interfere without the knowledge and concurrence of the occupier or manager with the means provided for the removal of fumes.

H. J. Gladstone,
One of His Majesty's Principal
Secretaries of State.

Home Office, Whitehall,
20th June, 1908.

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SECTION III

CARBON BISULPHIDE POISONING

BY E. R. HAYHURST, M. D., Columbus, Ohio.

Historical Notes.—Bisulphide of carbon was discovered by Lampadius, the German chemist, in 1796. Pazen[1] seems to have been the first to call attention to it as an occupational poison (1851) and to have counselled caution in its use. Duchenne[2] (1853) mentions its paralyzing effects. Delpech[3] (1856, 1863), with description of cases, gave the first comprehensive picture of its symptomatology. Tavera[4] (1865) first studied the blood changes. Since these dates much experimental work and the study of special signs of intoxication have been reported by various investigators.

Chemistry.—Bisulphide of carbon is a constituent of coal tar. It is made commercially when red hot carbon, contained in a retort, is treated with sulphur vapors. It is an endothermic compound and, therefore, formed only on heating. The carbon bisulphide gas is condensed to liquid form, purified and, when pure, is practically colorless, has a chloroform-like odor, is very limpid, mobile, volatile, refractive, insoluble in water, but soluble in all proportions in alcohol and ether and to 60 per cent. in oil. It dissolves all resins except shellac.[22] It does not dissolve vulcanized rubber. It dissolves nearly its own weight of sulphur at 38°C. When impure it has a repulsive smell and a yellow color. Impurities are usually H_2S, sulphur, and sulphydrates of carbons. H_2S cannot be demonstrated in its vapors,[10] at least in sufficient quantity to produce poisoning.[17]

When passed through a red hot tube, with chlorine, it yields carbon tetrachloride (CCl_4) and sulphur monochloride (S_2Cl_2), two substances much used as rubber solvents. Carbon bisulphide is more inflammable than ether and burns with a bluish flame yielding CO_2 and SO_2. In America it is practically all made by the Taylor electric-process, Penyan, New York, and is a rather cheap substance, averaging from 8 to 12 cts. per pound. It has a specific gravity of 1.262 and boils at the low temperature of 46.5°C. Its vapor is heavy; density 2.6. If evaporated rapidly, as by passing a current of air through it, its temperature is reduced to -60°C.; hence it may be employed to produce low temperatures.

Uses.—According to Molinari,[26] besides its principal use of vulcanizing and dissolving rubber,

> "Attempts have been made to use it for the extraction of sulphur from poor ores. It is used for extracting aromatic oils from drugs. It is also used in large quantities for the extraction of oil from seeds and from oily seed residues and rags. It is also used for degreasing vegetable and animal resi-

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dues, such as rags, bones, meat, hides, wool, etc. It is used for preparing solutions of wax for coating plaster casts, and for the preparation of waxed paper. It is also used for dissolving out the tar from many industrial products [and in gas purification to remove sulphur],[27] for preparing various chemical products, for making Greek fire (phosphorus dissolved in CS_2), for killing insect pests, and more especially, in large quantities, for destroying phylloxera in vineyards by injecting it into the earth with suitable pumps. . . . In 1906 it was used with good results for destroying the nematodes which attack beet root."

It is used in the preparation of cellulose for artificial silk; in cleaning establishments to remove grease spots; as a disinfectant; and by the Public Health Service in destroying rodents in their natural haunts.[28]

In the Parke's process of vulcanizing it is used to carry the sulphur into the rubber "in the cold." It is practically never used pure, but combined with S_2Cl_2, CCl_4 or with benzol (C_6H_6). It renders rubber more elastic, malleable, resistant and long-lived. It is a better solvent for rubber than benzine and even better than ether, and is likely to be the principal solvent used in all rubber and gutta-percha cements which are to dry quickly.

It has been advocated for the treatment of tuberculosis (all forms) by hypodermic injections and by inhalations of the vapor.[15]

Lehmann[10] has shown that 1 part per million in the air will produce mild symptoms, while 1½ parts will produce severe symptoms. As much as 2 to 3 parts per million have been found in work places.

Classes of Workers Most Liable to Poisoning.—(1) Rubber workers; (2) oil and grease extractors; (3) those using cements dissolved in CS_2; and (4), rarely, those concerned with its manufacture and various other uses mentioned.

Predisposing Factors in Industry.—Among these are hot weather or overheated rooms, also working in low places and close to the floor. Poisoning occurs through breathing the vapors, or getting the fluid on the skin, or having it splashed upon the clothing. Good ventilation in recent times has greatly decreased cases and usually only milder symptoms show.[16] In Belgium, Galet[18] says it causes "le plus grand" per cent. of injuries and incapacities for work. Repairers of belts in factories who use cement dissolved in CS_2 are affected, Briau[25] finding as many as 8 of 30 to be mentally "off." It is the cause of "folie du cuir" or leather-workers' insanity. It is used by shoemakers in applying invisible patches. Shoemakers may be very easily poisoned because of their close quarters, sitting near the floor, lack of precautions and ignorance of the poisonous character of the gutta-percha solvent used. The most susceptible persons are the ill-nourished, neurotic, alcoholic, female and young persons.

In Ohio where over 30,000 workers were found to be employed in the rubber industry (1914), less than 1 per cent. came into contact with CS_2. The classes usually exposed were: (1) Rubber cement makers (exposure usually quite limited since the mixing is mechanically done in closed up

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containers). (2) Cement tube- and can-fillers (more exposure, but it was found that boys and men so engaged were a rapidly changing lot or rotated with other work. Mild symptoms were a frequent complaint, however). (3) "Cold curing" of drug sundries (gloves, caps, nipples, bags, cots, etc.), and splicing ends of inner tubes for tires (exposures varied in plants, but some form of local exhaust ventilations was usually present, or a more or less complete confinement of the odors within cupboards, or, finally CCl_4 was largely substituted for CS_2. Some moderately severe cases were found). (4) Transmission belt makers. Balata is the kind of rubber usually employed (exposure varied but quarters were spacious and not many employees thus engaged. Reports of acute cases in the past were all that could be found). (5) Water-proofing and spreading of rubber onto fabrics (CS_2 used very limitedly, if at all, for this purpose, since benzine seems to be the universal solvent). In almost no processes were females exposed to CS_2.

Pharmacology and Animal Experiments.—Oliver[21] found that CS_2 was a very deadly poison to animals, and a more powerful anæsthetic than chloroform and more lasting in its effects. A rabbit which inhaled the vapor for a short time showed intense excitement, intoxication, impulsion, followed by a deep sleep, and on regaining consciousness its hind limbs were paralyzed and so remained for 1 hour. Animals exposed more slowly died within 3 to 21 days, during which time they lost flesh, showed tremors, were easily fatigued, became paralyzed in the hind limbs and died convulsed. The urine was excessive, no albumin was present, there was but little urea, although considerable sugar at death.

The cells of the motor areas of the cortex showed, by Golgi's method, cytoplasm stained unequally and axis-cylinders distorted. Oliver states that it is a fairly safe anæsthetic, but the odor is too repulsive for use and it produces too much muscular jactation. Red cells are not disintegrated (dog and rabbit); spectroscopic findings are all negative.

There occur extravasations in the lungs (Poincaré[5]) and stomach walls;[28] pneumonia,[18] degeneration of liver cells, of splenic pulp, and sometimes nephritis, and gastro-enteritis. The myelin substance of the nervous system is broken up into fatty droplets,[5] and emboli occur in the blood-vessels of the brain. The blood findings in animals have been well investigated and do not differ materially from those in man (see under Pathology).

Pathology.—Persons poisoned by bisulphide of carbon, in the course of conducting occupational processes, usually show atrophy and fatty degeneration of muscular and connective tissues with loss of normal fat. There may be dryness and contraction of the skin due to the fatty solvent action of CS_2 (Perrin).[27] The urine in acute cases may give a dark-brownish color (Galet) with Fehling's solution, due to the presence of hematin from destroyed red cells. Hydrobilirubin and indican, as well as albumin and casts occur in older cases. Its essential action on the blood is hemolysis. Cenci[20] carefully studied 12 afflicted workers who had been exposed for long periods,

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while engaged in the extraction of oil from olives, and obtained blood findings as follows: (1) chloranæmia, (2) hypoglobulia, (3) white cells not affected, (4) alkalinity of the blood reduced in all cases, (5) red corpuscle resistance greatly increased, (6) no apparent effect upon coagulation time. All of these conditions returned to normal after withdrawal from the work and within a period of a few weeks or months. Jump and Cruice[14] report hemoglobin 40 per cent. with red and white cell count normal. Galet[18] found no granulations in the cells in either acute or chronic cases in six men. Laboulbéne[6] was the first to cite cases showing cutaneous "taches ecchymotiques," later followed by pigmentation. These appeared upon the limbs and thorax. Vesicles may appear.

Symptomatology.—Two types of industrial cases are recognized, acute and chronic, the latter with two stages, excitement and depression. Acute industrial poisoning is rare to-day. Extensive users of the substance appear to be well informed of its dangers. However, in Ohio in the first 6 months of the year 1914, two instances came to our notice, which may have been due to carbon bisulphide, in one of which sudden paralytic attacks occurred on each of two times the patient returned to work, while, in the other, puzzling symptoms, which lasted a few days, were followed by death with hemorrhages from the various orifices. In rubber workers symptoms of carbon bisulphide poisoning are apt to be accompanied by those due to benzine, benzol, sulphur monochloride and carbon tetrachloride, which are usually employed, one or all, with the bisulphide.

The acute cases have as chief symptoms: (1) circulatory disturbances, pallor, headaches, throbbing of temples, pains in nape of neck and crown radiating to temples and frontal region, palpitation, fainting; (2) drowsiness; (3) digestive disturbances, especially nausea, anorexia, repeated vomiting, perhaps diarrhea, but sometimes colic and constipation; (4) nervous manifestations, especially incoördination, weakness in legs, signs of great fatigue, unsteady gait, impulsive acts, as throwing one's self under vehicles, into machinery, out of windows, etc.—mania with homicidal and suicidal results are known. There are also marked hallucinations of sight, hearing, taste and smell. Death may follow during a convulsion or during coma. The breath has the fetid odor of sulphides.

Chronic symptoms are the usual ones and the onset may appear within a few weeks or months, or only after years in the work.[24] The stage of excitement comes first, and is usually evinced while at work as exhilaration, loquaciousness and undue jollity. Following this comes faintness, giddiness, drowsiness, exhaustion and headache. At home the patient is irritable, depressed, tastes the bisulphide in everything, sleeps in daytime, cannot sleep at night, and has bad dreams. Return to work temporarily invigorates again. Cases investigated in Ohio complained of feeling "drunk," when fellow-workers would take them into the open air (with little concern). Usually new men were the ones most afflicted. Hysterical manifestations[7]

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are particularly common, and more so in females. There are all manner of psychical phenomena, from "crazy," excited talk, to ravings and acute mania. Workers complain of dysthesias, which assume geometrical distributions; numbness and tingling; anæsthesia of parts; "that contact of the hand with the body seems that of some other person's;"[18] cramp-like pains; spasms of face muscles; and trembling. There is a drunken gait, exaggerated reflexes, and a Romberg sign is usually present. A violent explosive temper with symptoms resembling delirium tremens[21] may prove puzzling in diagnosis. Peripheral neuritis, mostly motor in type, is easily demonstrable. Digestive disturbances include vomiting, especially in females, loss of taste, subjective rotton odors, and diarrhea, which may alternate with constipation. Delpech[3] cites an instance where a capricious appetite showed that "he spent 10 francs to each 6 sous' worth." The pulse is rapid and there may be abundant nose bleed at times. Menorrhagia and miscarriages occur, while a violent sexual passion possesses males.[25] Hearing is early impaired, and more often than sight. Disturbances of sight[11] come later on—dimness of vision, blind spots, disturbances of color fields—and appear to be a retrobulbar neuritis with changes, later, in the retina and choroid.[19]

In the stage of depression, which follows the above in the course of a few weeks, there is noticeable nutritive disturbances, atrophy and emaciation, or sometimes just softness of the muscles and tissues, with fibrillations and relaxation. Pallor and weakness may develop into actual cachexia. Functional troubles are very common, and include paræsthesias (itching, formications), great depression, weakness of memory for words, puerile enunciation, melancholia, hypochondria and torpor. Organic nervous symptoms are almost equally common, and consist of partial and complete paralyses, especially of extensor groups of muscles (arms, hands, feet), leading to wrist drop, steppage gait, and, later, contractures. The fingers which come into contact with carbon bisulphide are apt to become extended, stiff and numb.[3] There is often tremor of the extended hands and tongue, exaggerated on use. The lips tremble, and quite violently so, when attempting to whistle.[14] Absent knee-jerks and other deep reflexes, with loss of sensations caused Berbes (1885) to speak of the condition as pseudo-tabes.[16] There is constipation all the time. Skin pigmentation may be present. The patient cannot read because of dim vision; fingers may, with difficulty be counted.[12] Pupils appear normal, there is no diplopia; the examination of the fundus shows, at first, a congested disc with hyperæmia of retinal vessels, and, later, pallor, said to be more marked in the temporal half. Deafness may be marked. Sexual power is entirely lost; atrophy of testes may be plainly discernable. Sterility obtains in females. Ten cases (all males) discovered among the rubber workers in Ohio, lay between the excitable stage and this chronic depressive type, with some symptoms especially well-marked.

Diagnosis.—Carbon bisulphide gives no characteristic sign or syndrome. Peripheral neuritis, hysterical symptoms, and history of exposure usually

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suffice for diagnosis. Physicians in rubber districts are so confused with the multiplicity of poisons used in that industry that they usually style all patients "rubber poisoned." An attempt is here made to distinguish between them. Acute cases are to be distinguished from poisoning due to: (*a*) Anilin—sudden weakness of legs, sudden swooning, cyanosis of lips, pale or suffused face, air-hunger, strangury, bloody urine, methemoglobin, stupor, apt to continue for 24 hours or longer, and, if death does not occur, bladder irritation and skin eruptions may follow. (*b*) Benzine —usually begins with foolish prattle, laughter, shouting, trembling and jerking—"the benzine jag"—collapse, pallor changing to cyanosis, syncope, all with quick recovery in fresh air. (*c*) Benzol—is more of an irritant, shows slight flushing of face, followed by pallor, watery eyes, nasal discharge, coughing. When unconscious, lips are of scarlet hue, blood is bright red and thin, tonic and clonic spasms are present; symtpons are much worse and more prolonged than those due to benzine. (*d*) Sulphur monochloride—harmful only when it is broken up by moisture (steam) into SO_2 and HCl vapor. (*e*) Carbon tetrachloride—rarely poisonous industrially, but will produce symptoms similar to chloroform, with nausea, coughing, headache and pronounced excitement. Around tar and gas works: (*f*) Hydrogen sulphide—an irritant, producing watery eyes, running nose, coughing, herpes on lips, contracted pupils, slow pulse, Cheynes-Stokes respiration, tonic spasms, delirium and coma; breath smells of sulphides, also. (*g*) Carbon monoxide and illuminating gas poisoning—may, rarely, be confused with carbon bisulphide poisoning. Finally, (*h*) Acute alcoholism—the patient is rather less impulsive, and there is a different odor to breath; unconsciousness is not coma.

Chronic poisoning shows itself principally in the history of the case. A period of several days or weeks of drunken jollity and excitement, followed by depression, with paralytic, digestive and nutritive disturbances, is quite characteristic. The condition is to be differentiated from: (*a*) Lead poisoning, which is more common in rubber-tire and hard-rubber factories than bisulphide poisoning. Here the predominance of colic, constipation, vomiting, with peculiar pallor and blue line on gums, and lead in urine are diagnostic. (*b*) Mercurialism—trembling, salivation, gingivitis, diarrhea, and foul breath are characteristic. (*c*) Duchenne[2] declared that time only would separate bisulphide poisoning from paresis, since death is not frequent in the former. This condition and (*d*) locomotor ataxia, however, have characteristic pupil, reflex bladder and rectal disturbances and specific blood and spinal fluid tests. (*e*) Progressive muscular atrophy presents no digestive disturbances, while mental faculties remain good. (*f*) Hysteria probably cannot be differentiated symptomatically, but note especially signs of peripheral neuritis of toxic origin. (*g*) Many mild cases probably pass as neurasthenia. (*h*) Chronic alcoholism with neuritis exhibits cutaneous and muscular tenderness, features usually absent in CS_2 poisoning. Also eye symptoms are not prominent. (*i*) The dulled mentality, muscular

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weakness and slow movements, which accompany or follow chronic gas poisoning must be thought of.

Prognosis.—Of the eight cases studied by Briau,[25] two had mental alienation, one had presenile involuntaries, one was profoundly stupid, three suffered paresis of legs, with pains in limbs, and one, brutal genital excitation. Except the first three, all recovered, with the possible exception of full capacity for work. Delpech[3] says, "He who has worked in sulphur is never again a man." Usually cases lightly affected, or only acutely affected, and who get out of the work, are not harmed. Chronic poisoning rarely results in death, except through cachexia. Eye symptoms usually recover, hearing recovers more slowly, while memory for words seems to be one of the last functions to be reëstablished.

Peterson[8] reports recovery in three cases of mania within 9 to 17 months. Bard[9] reports cases of homicide and suicide, and another case which recovered from mania.

Treatment.—Prophylaxis is usually easy. Workers should stand on raised platforms, not down in pits or low places. They should be selected, instructed, alternated and allowed only short applications at this work. They should wear impervious clothing, rubber gloves, and boots. Local exhaust systems; slatted floors with powerful exhausts beneath; fresh air from windows stirred up by electric fans; the process confined within special cupboards with apparatus for subsequently drying the goods, all handled mechanically—these are some of the various means employed to prevent poisoning. It should be remembered that clothes, hands, walls, floors, etc., become impregnated with CS_2 and that all need thorough and frequent cleaning. Especially should the hands and fingers be kept out of CS_2 solutions.

Carbon tetrachloride is the natural substitute, having many advantages, but is more expensive. Yet it is extensively used in Ohio. Some patent harmless substitutes are also on the market.[22] Substitution of other curing processes (steam, vapor cures with sulphur monochloride alone, sulphur chloride with carbon tetrachloride, etc.) have largely replaced the old "acid" or "cold" cure in rubber districts.

In cases of acute poisoning, remove to fresh air, give warm bath or cold affusions, accordingly, as excitement or stupor prevail. The pulmotor may be necessary. For subacute or chronic symptoms the patient should quit the work at once. He should be given nitrogenous food to prevent muscular atrophy, with such drugs as strychnine, quinine, phosphorus, iron, iodides and arsenic. For the muscular conditions, electricity (galvanism), vapor baths and massage are indicated.

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SECTION IV

CARBON MONOXIDE POISONING

BY G. L. APPELBACH, M. D., Chicago, Ill.

The subject of carbon monoxide poisoning is an intricate one. It is not difficult to find that workers in certain industries are being "gassed," either by appreciable quantities or by small continuous doses of this gas. It is, however, difficult to determine the sequelæ or subsequent effects of carbon monoxide gassing. Many factors make a scientific conclusion of the effects of carbon monoxide gas prohibitive. These are syphilis, alcoholism, poor nourishment, insanitary home conditions and surroundings, and the element of fatigue and high temperatures. These factors had to be considered in the investigation of 1910 by the Illinois Commission on Industrial Diseases, and are likewise reflected in this chapter.

Acute carbon monoxide poisoning is that condition in which the person affected has been subjected to such a toxic dose of the gas as to produce either coma or such a train of subjective and objective findings as to be readily attributed to "gassing." Chronic carbon monoxide poisoning results in the worker's being subjected to repeated or continuous small doses of carbon monoxide gas, not, however, producing at any one time a marked symptom-complex.

Before going into the industrial medical importance of this toxic agent, we must pause to consider with what temerity we use this most poisonous gas in our everyday life. During childhood the writer became acquainted with the subject of carbon monoxide poisoning when the kindly grandmother warned her grandchildren to keep the stove door closed, especially at such times during which the blue flames skipped about, between and on the surface of the burning coals. We permit our houses, even our sleeping rooms, to be surrounded by a network of pipes containing this deadly gas, making us the victims of accidental leakages. Our illuminating gases vary in carbon monoxide content, the toxic agent of the gas. Carburetted water gas is used extensively throughout the United States for illuminating purposes with contents of about 30 per cent. carbon monoxide. Since 0.5 per cent. to 1.0 per cent. of carbon monoxide or 1.5 per cent. to 3.0 per cent. of illuminating water gas is rapidly fatal, accidental leakage of this gas in our bedrooms often causes death. Accidental "gassing" was more common during the introductory period of illuminating gas than at present. Many cases have also occurred from neglect of closing stove doors, from back draught of hearths or fireplaces, from old-style bake ovens and charcoal braziers. The use of any form of fuel, such as charcoal, wood, coal, kerosene and gas, is

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dangerous in any room without an adequate method of drawing off the products of combustion. A case in point was recently reported to the writer. A young man was overcome by carbon monoxide while taking his bath, the room being heated by a portable gas stove in which the carbon monoxide formed at the cooling surfaces of the stove.

Police records of almost every city show numerous cases of gas poisoning, often proving fatal, attributed to intentional suicide. The Paris records, for the 9 years from 1834 to 1843, show a total of 4595 deaths due to suicide, of which 1432 were accomplished by means of carbon monoxide. The charcoal brazier became the popular means of suicide in France, so much so that we find frequent reference to it even by writers of fiction such as Dumas, Victor Hugo and Eugene Sue. With each decade this practice increased, until the records of 1891 in Paris disclose the surprisingly large number of 848 suicides by this method.

Additional accidental poisonings from carbon monoxide gas, other than industrial, have occurred in many different ways, chiefly from fires made with briquettes, used in heating cabs; from chemical laboratories; among persons living in the neighborhood of gas-producing industries; from gasoline automobiles and launches. Reference is made to the numerous stories about tramps expiring in the warmth of a brick or lime kiln. This gas is often formed by smouldering timbers after large conflagrations in factories, theatres, and other buildings. The writer remembers reading about chemical analyses of air upon the discharge of German field artillery using smokeless powder. The examination of this air disclosed the fact that 30 per cent. CO was present. Surgeon General Stokes, U. S. N. finds that the pulse rate of men rises rapidly from 72 to 120 within 10 minutes after firing guns in the turrets on battleships. He also observes that the so-called "heat prostrations" in the stoke rooms are often due to carbon monoxide and other gases.

CHEMISTRY

Carbon monoxide is seldom found in a free state, except in the chemical laboratory and in old gas mains where it lies inert in brick, flue dirt, soot, oil, and pools of water. The gas is colorless, tasteless, and without odor, although some writers claim its odor to resemble that of garlic. It burns with a blue flame, but does not support combustion. It has a specific gravity of 0.967. One liter weighs 1.25 grains. It is very diffusible and readily held inert by dust and oils. Claim has been made that it is able to diffuse through red hot iron; however, such proof is lacking in our experience. It is readily taken up by CuCl.

$$Cu_3Cl_3 \text{ plus } 2CO = Cu_3Cl_2.3CO.4H_3O$$

This reaction is commonly used in gas analysis for removing the volume of carbon monoxide.

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A good qualitative and quantitative method for detecting and measuring the carbon monoxide content of a gas is to pass the gas through KOH which removes CO_2 and then through I_2O_5, which liberates iodine. The iodine is recovered in a starch solution, allowing the determination of CO. The following reaction occurs:

$$I_2O_5 \text{ plus } 5Co \rightarrow 5CO_2 \text{ plus } I_2$$

Carbon monoxide gas is most frequently the result of incomplete combustion of carbonaceous materials. Wherever combustion of carbon-containing material occurs, we find CO as a by-product. Complete combustion is the rarer occurrence, as observed in our furnaces and stoves. The amount of CO formed, however, may not be perceptibly noticeable. Even the smoking of a cigar or cigarette is accompanied by the formation of CO gas. Combustion depends on the presence of oxygen, in a practical way on the draft or on weather conditions. When a flame strikes a cooling surface, such as a metallic plate, combustion may also be incomplete. The complete combustion of carbonaceous material results in CO_2, the incomplete in CO plus other by-products.

CO may also be formed by the de-oxidation of CO_2, when the latter is passed over red hot coals or metal. CO formed by incomplete combustion is found in our stoves between and beneath the coals; when formed by deoxidation, on the surface of the red hot coals.

CO is also found in the after-damp of mine explosions, its presence being determined by the amount of methane which has undergone combustion. If this gas is present in volumes over 9.5 per cent., CO is formed in the after-damp.

Fortunately CO is usually in composition with other gases, such as CO_2, hydrogen, methane and illuminants, some of them imparting an odor making the presence of CO detectable. The composition of these various gases, as used industrially, will be referred to later.

TOXICOLOGY

From the view point of the industrial hygienist, or occupational disease physician, CO is the most important toxic gas, being not only highly toxic, but used extensively in our various industries. Gruber[1] says that the limit of toxicity is 0.02 per cent. A volume of air containing 0.01 per cent. may cause distress, headache, nausea, and other phenomena. Others say that symptoms will not be produced until 0.05 per cent. of the gas is present. The point of toxicity will vary in individuals. This has been accurately proved by Haldane[2] in humans and animals. From experiments made by Sir Humphrey Davy it is recorded that about 0.08 gram (about 12 grains) of CO is fatal to a man of 154 lb. weight (Peterson and Haines). The blood is capable of carrying 1 liter of oxygen or 1 of CO. As a matter of fact such

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will never occur, since we never find a saturation of the hemoglobin over 83 per cent. in fatal cases. It is well known that an air volume containing 1.0 per cent. of CO is considered as having moderately rapid fatal tendencies. In the Snaefell Mine Disaster,[3] Dr. J. S. Haldane and his co-workers observed that the mine air contained 1.07 per cent. CO.

The gas acts rapidly with no irritation to the respiratory tract, and also because of its inodorous quality its presence if often unknown. A case is recorded by Sonnenschein of a chemist who, by a single inhalation of an atmosphere laden with the gas, fell backward as if struck by lightning.

CO is more toxic for certain animals than for others. Birds die instantly in an atmosphere containing 5 per cent. CO. It is said that mice are still more susceptible to the gas. If CO is present in toxic doses it will affect mice sooner than humans. Haldane[2] has made use of this information, applying it to safety practice in the mining industry. This is a cheap and simple safety precaution which can be made use of in our mines.

An interesting case is reported by Jeffries[4] of a person and a linnet who were overcome by illuminating gas due to an accidental leakage. Of importance is the fact that the human was found in coma and recovered, whereas the bird was found dead.

The effect of CO gas varies in animals, causing convulsions in rats and other animals, while birds die failing to show such signs. Incidentally, the writer might make mention here of the method of killing vagrant dogs in Chicago by this gas. The writer has on several occasions killed guinea-pigs by thrusting them under a bell jar full of illuminating gas. It took but ½ minute for the pigs to topple over, followed by general convulsions and death in from 2 to 3 minutes.

The gas is toxic to the human whether it is inspired, swallowed or injected into the peritoneal cavity. The CO rapidly unites itself with the hemoglobin, replacing the oxygen and forming a very stable compound known as carboxyhemoglobin. The affinity of the hemoglobin for CO is 300 times greater than the affinity for oxygen (Haldane). This substitution of CO for oxygen deprives the blood corpuscle of its oxygen-carrying power.

There is a difference of opinion as to whether CO merely causes internal asphyxia by reason of having deprived the blood corpuscle of the hemoglobin element and, therefore, its oxygen-carrying power, or whether the gas also has a distinct toxic action on tissue.

Haldane refers all toxicity of the gas to the lost oxygen carrying power of the hemoglobin, pointing out that a saturation of 20 per cent. will cause dizziness and dyspnœa, 50 per cent. loss of consciousness, 80 per cent. death. He points out that such is due to an internal asphyxia and explains that even such pathological sequelæ, as fatty degeneration of vessels and other organs, are due to the impoverished red blood corpuscle. To sustain his point he proves by experiment that animals will live much longer and

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will tolerate a greater quantity of carbon monoxide in an atmosphere of oxygen than of air.

However convincing Haldane may be, we must consider the question whether there is some other toxic value in carbon monoxide. Following CO poisoning, even with small doses, sequelæ frequently occur, particularly in the nervous system, in which there are definite pathological changes. Moreover, the gas is rapidly narcotizing, and the earliest symptoms, such as the weakness in the lower limbs, are strongly suggestive of some distinct toxic action. The latter opinion of the toxic action of CO is strongly supported by Gruber and Kobert.[5] The writer attempted to kill guinea-pigs in equal volumes of CO and of CO_2 and found, of course, that the animals died much more rapidly in CO than in the CO_2. But the error in this experiment can be readily seen, since it is evident that the oxygen-carrying power of the blood could have been much more rapidly depleted by one gas than by the other.

The question of toxicity of this gas is an interesting one, but we must leave the argument in the hands of the toxicologist. The writer has always leaned to the opinion that CO has a definite toxic action on tissue, but recently, owing to the work done by Haldane and, also, because of the work done by Dr. Matthew Karasek and the writer, in which it was demonstrated that humans subjected continuously to this gas had a compensatory polycythemia and no physical disturbances, the writer has inclined more and more to Haldane's views.

The CO exactly displaces the oxygen of the hemoglobin, forming a very stable compound which is probably never disassociated until the blood corpuscle meets its death and is eliminated.

ACUTE CARBON MONOXIDE POISONING

Acute carbon monoxide poisoning is found in many industries, the nature of which will be dwelt upon in detail. In previous work on industrial "gassing" reference has been made to nearly all the industries in which acute gassings" occur, but the special points of danger as existing in each have never been clearly brought out.

Following is a table of statistics gathered from the reports of the Chief Inspector of Factories and Workshops, England:

Year	CO gas poisoning										Sulphuretted H		CO_2		Cl		Nitrous fumes		Dinitro benzol		Benzin		Other	
	Total		Blast furnace		Power		Coal		Other															
	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal	Non-fatal	Fatal
1908	49	6	23	3	17	2	8	1	1	0	7	1	1	4	1	0	2	1	2	0	2	0	3	0
1909	37	6	6	0	21	4	10	1	0	1	3	2	0	2	1	0	10	2	4	0	0	1	4	0
1910	44	9	12	7	25	0	4	0	3	2	2	0	1	1	2	0	11	0	18	0	0	0	4	0
1911	58	6	14	2	30	1	4	2	10	1	6	2	0	1	5	0	16	2	21	0	21	0	0	4
1912	77	14	28	5	15	4	27	2	7	3	6	0	1	2	3	0	11	1	9	0	2	1	1	1

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This table shows that gases containing carbon monoxide cause more industrial sicknesses and deaths than the other gases referred to. The greater number of CO "gassing" cases occurred from producer and blast-furnace gas. Those not thoroughly acquainted with the subject are confused when such a variety of gases are mentioned as coal gas, producer gas, water gas, blast-furnace gas, power, suction, Monde, and other gases. It should be constantly kept in mind that they all contain carbon monoxide in varying quantities, and that it has been fairly well established that carbon monoxide is the toxic agent in all these gases. Their toxicity will vary because of the differences of CO content, diffusibility, and also because of their different methods of production. It seems that the above tabulation cannot include cases of CO poisoning which have occurred in coal and other mines. Following is a tabulation of the industries in which carbon monoxide poisoning has been observed.

The manufacture of steel:

1. From blast-furnace gas.
2. In the engine houses and boiler rooms and where blast-furnace gas is used as fuel.
3. From producer gas.

The use of power or producer gas:

1. Smelters of zinc and other ores.
2. Tin can manufacturers.
3. Foundries.
4. Glass bottle manufacturers.
5. Coke by-product concerns.
6. Chemical companies.
7. Laundries.
8. Tailoring concerns.
9. Letter press printing.
10. Laquering stoves.
11. Book binderies.
12. Box making.
13. Metallurgical plants.

The manufacture of illuminating gas:

1. Carburetted water gas.
2. Coal gas.

Industries using ovens:

1. Smelters.
2. Brick kilns.
3. Cement kilns.
4. Bakeries.
5. Enameling of porcelain.
6. Miscellaneous.

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Heating and steam power plants:
1. In the engine rooms of large vessels.
2. Fire and boiler rooms in all industries.

Where "gassing" occurs with moderately ill effects:
1. The use of salamanders in drying newly plastered and painted buildings.
2. Electrotypers.
3. Stereotype, monotype and linotype rooms.
4. Canneries.
5. By the use of the tailor's goose.
6. Metal-casting machines.
7. Heating, soldering and other metal pots.
8. Mining—in explosions.

From the fumes of gasoline engines:
1. Garages.

Where CO is used in preserving fruit and vegetables:
1. Freight cars.

Industries not mentioned can be readily classified in above scheme of classification.

THE STEEL INDUSTRY

The blast-furnace gas as produced in the average American stack will run an average composition as follows:

CO	26.0 per cent.	CH_4	1.0 per cent.
CO_2	11.12 per cent.	N	57.0 per cent.
H	3.5 per cent.	O	0.2 per cent.
C_2H_2	0.3 per cent.		

This gas is produced in the blast furnace by the smelting of iron ore, coke and limestone, in the proportion of 2000 lb. of ore, 2000 lb. of coke, and 800 lb. of limestone. After combustion has begun, the mixture is blasted by means of heated air, which is heated in the adjoining stoves, four to one furnace. The air in the stoves is heated by means of blast-furnace gas. The gas which is produced in the blast furnace goes up the stack to the top from where it is led by a pipe through a dry cleaner or dust collector. From here it goes to the gas washer, which allows the gas to ascend through screens of dropping water, removing thereby that dirt not caught in the dry cleaner. It is then piped in an overhead main to two points, the stoves, and the boiler and engine houses. There are four stoves for each furnace, two being used only at one time. In these the gas derived from the blast furnace undergoes combustion, heating the air which is also drawn through the stoves. That portion of the gas which is piped either to the boiler houses or to the gas engines, goes through an overhead main to its destination. Some steel companies use the gas in boiler houses to produce steam; others

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explode it in gas engines. Both methods produce power, wherewith the converters, rail mills and other machinery is operated.

Changes in the operation of the blast furnaces are constantly occurring. A new method of gas cleaning is being adopted, which will reduce the risk of gas poisoning. Instead of using a dry cleaner which needs a daily attention, the gas will be treated by washing, which process is not so productive of gas poisoning.

There are certain places along the gas route where "gassing" is liable to occur.

The tops of the furnaces have always been considered notoriously dangerous. This is not so true to-day as it was 10 and 15 years ago. The modern blast furnace has an automatic filler. The ore goes up a cable hoist and is dumped into a bell valve. Formerly three to four men were constantly required at the top of a furnace. At present only "cleaners" or "sweepers," "sailors" and "riggers," needed in urgent repair work, are required to ascend to the top while the furnace is in operation, and then only for 20 minutes every 12 hours. Before going up on a furnace, the foreman is notified and attends to all proper safety precautions. No man is ever allowed alone on the furnace.

The "mantle" above the bustle pipe is nearly as dangerous as the top. It seems impossible to prevent leakage of gas through the walls of the furnace, which generally occurs in a belt around the furnace about 12 ft. from the ground, including the area called the mantle or platform, running around the furnace. Here the gas is found frequently in sufficient quantities to maintain continuous combustion when ignited. On the other hand, if the gas remains unignited, especially on rainy days, it affects those on the casting floor.

The great danger about the stoves is at the enormous burners from adverse draft or puff-back of unconsumed gas and also from leakage. A special danger exists during the cleaning out of a stove through one opening while the gas is burning at the other.

Another source of "gassing" is in cleaning out cold mains or pipes, because the gas remains inert in the flue dust and is liberated in the atmosphere upon being disturbed. This is done less frequently at present, because of newer methods of flushing the mains with water. In the old-style furnaces, where the mains were underground, cleaning was a very dangerous operation. A series of pipes, called boot-legs, descending from the overhead main on its way to the engine and boiler houses, is another source of "gassing" during cleaning time.

In the boiler house the same danger of puff-back is present as at the stoves, except in the later make of boilers, which have the entering flue burners sealed into the brickwork, and receive air from a point about 2 ft., below. This type of boiler we saw only in the Illinois Steel Company's alternating-current boiler house. With this kind of furnace an explosion

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may occur, if the gas is carelessly turned on too rapidly at first. During the investigations many flues with large leaks or rents were noticed, either discharging the gas under pressure into the boiler room, or plugged indifferently with clay stoppers. Cleaning out the boilers frequently gives rise to "gassing," both in the removal of débris from the fire-box and space, and from the boiler drum.

The dry cleaner is opened daily. "Gassing" may also occur from this procedure.

Accidental leakage usually occurs in the basement of the gas-engine house. The men who work above the engines, such as oilers, often complain of headache, especially after having worked above the engines for a prolonged period. In one steel mill an engineer was found dead on the bed of an engine. The coroner's verdict was CO poisoning.

It is relevant to state here that the experience of the writer and the reports of the English Factory Inspector coincide in regard to the usual occurrence of "gassing" in the steel mills. A great many occur during cleaning operations, when the furnace and mains are not in operation. Even after air has been driven through these mains, CO lying inert in the flue dirt is liberated when cleaning operations are conducted. From a review of the English Factory Inspector's reports, it can be seen that out of 68 cases of carbon monoxide poisoning which occurred in blast furnace work, 23 cases representing 33 per cent. of the total number occurred during cleaning cold mains or furnaces.

The most exposed to blast-furnace gas are cleaners of cold mains, repair men such as "riggers" or "sailors," oilers, and sweepers.

Since "gassing" may occur anywhere along the gas route, we will next mention other less common sources in the regular routine of furnace operation. Structural iron men have been "gassed," while rigging up a smoke stack over an open-hearth shed or building, or while working on buildings in the vicinity of the furnace. Men in supposedly safe places are sometimes overcome by vagrant whiffs of gas due to peculiar atmospheric conditions. Men have taken stolen naps alongside the blast-furnace stoves with fatal consequences. Then there occur freak "gassings" which even the most expert safety man would never anticipate.

The majority of steel mills, while obtaining most of their gas from the blast furnaces, find it necessary to operate one or more producer gas furnaces for the purpose of supplying power to such parts of their plant where blast-furnace gas is not available. Most of these furnaces being located in the open are, therefore, seldom the cause of acute CO poisoning.

In this connection it is well to state that "gassing" is not only the cause of such disturbances as drowsiness, coma, and other signs of gas poisoning, but is also the cause of serious accidents, such as result from falls after having been overcome by gas, or from burns due to gas explosions. Burns resulting

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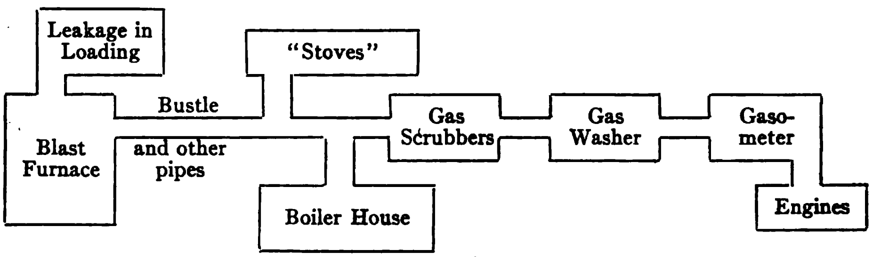
from explosions have occurred in and about gas stoves and in boiler houses of steel mills.

The following table illustrates the result of our investigation concerning the incidence of "gassing" and also the gas route:

Source of gas	No. of men employed	Fatal cases	Severe cases per year	Mild cases per year
Blast furnaces............	.900	13 during past 4 years	65	216
Boiler houses............	212		1	55 estimated from findings
Gas engine...............	42	1 in 1910	4	Variable
Open hearths............	24	....	1	Variable

GAS ROUTE



Gas poisoning and accidents due to gas in steel mills have in the past decade or two shown a decided decrease for the following reasons.

New and modern types of furnaces have been installed. Automatic charging of furnaces has eliminated those men who formerly were stationed at the tops of the furnaces. The modern furnace is of more substantial and heavier construction with a view of accommodating more plates and fire-brick. Overhead mains have replaced underground mains. Moreover, underground pits have been done away with. Safety organizations of the steel companies have adopted suitable safety rules, which incorporate the following ideas: No man is allowed upon any furnace alone; repairs must be undertaken by two or more men; a watcher must accompany every workman in any place of hazard; special placards and transportable danger signals are placed in the danger zone (see pictures exhibiting character of signs used).

ILLUMINATING GAS INDUSTRY

Two distinct types of gas are used for illuminating purposes, water and coal gas. Following is an average of tests made of the composition of water and coal gas:

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	Water gas (carburetted), per cent.	Coal gas, per cent.
CO_2	3.00	6–7.0
CO	30.00	0.0
Light oils	12.00	5.0
H_2	31.5	46.0
CH_4	18.0	40.0
O	0.5	0.5
N_2	4.5	2.0

The use of water gas is prohibited in many cities necessitating the use of coal gas. In certain cities of England the use of a gas containing over 14 per cent. of CO is forbidden for illuminating purposes. A review of gas poisoning shows a considerably greater number of "gassings" occurring in cities using water gas than where coal gas is used. The illuminating gas industries in Chicago employ about 7000 men, of which, however, only 2000 to 2500 are exposed to the gas. These men are engaged in operating the various stations where the gas is manufactured, in repairing of leakages in mains, and in construction of new mains.

In order to give a clear understanding of the dangers of "gassing" the process of manufacture is described here. A charge of coke is blasted in the generator, this part of the process being called the "blow." Steam is then blown in, forming what is then known as blue water gas, which has 43 per cent. CO. The gas is next carburetted by the addition of pre-heated gas oils, reducing the CO content to about 30 per cent. After being carburetted the resulting gas is passed through what is known as the superheater, which consists of a checker work of brick, heated so as to fix the constituents of the gas. From here the gas is led through a main to a condenser, which cools and to a certain extent mechanically cleans the gas. From there the gas passes to the shaving scrubbers, which removes most of the remaining tar. From here the gas is passed through large boxes, containing hydrated FeO_2, which removes sulphur compounds and all remaining tar. Having been purified, the gas is metered and passed into large tanks, a common sight in larger cities, where it is ready for distribution to the consumer.

Up to this point accidental leakages may occur, causing "gassing." Again the men are sometimes gassed while cleaning, especially about the generators, mains and oxide box.

Gas mains are often laid in our streets parallel to a sewer or an electric conduit. Therefore, "gassing" occurs not only to the men who work on the gas mains, but also to the municipal sewer and water pipe extension crews, and to members of telephone, telegraph, and electric lighting gangs. The writer has personally assisted in rescuing from a manhole two repair men who had been overcome by gas escaping from a defective main while repairing an electric connection.

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PRODUCER GAS

Producer or power gas is used in the industrial world to generate power by means of a gas engine, to be used as fuel in producing steam and electricity or for heating purposes. The industrial use of producer gas is increasing rapidly. Therefore, various types of this gas are manufactured. Any kind of carbonaceous material can be used for making producer gas. The most commonly used material is coal. In some types straight distillation occurs while in others steam is used as part of the manufacturing process. Thus the composition of the gas will vary depending upon the method of manufacture. According to some methods the gas is produced by suction, to others by blasting the air. Common names for producer gas are: power gas, suction gas, Monde gas, and wood gas. An average composition of producer gas is as follows:

	Per cent.
CO	23.0
CO_2	8.0
H	11.0
C_2H_4	0.2
CH_4	0.2
N	57.1
O	0.2

The English reports show a large number of "gassings" from producer gas. The accidents depend on the situation of the generator, on whether suction or pressure is used in making the gas, and on the general safety rules followed. A producer in an out-door location is naturally not as dangerous as one placed in an inclosure. Producer gas is used in metallurgical plants, by glass makers, for heating stereotype, linotype and monotype kettles, the iron in tailoring establishments for pressing clothes, in laundries, book binderies, smelters, laquering stoves, and numerous other fields of industry.

INDUSTRIES USING OVENS

Ovens are used in various trades for divers purposes, such as in the melting of ore, enameling of porcelain and plumbing supplies, and enamel ware, in bakeries, chemical companies, and in other industries. Carbon monoxide poisoning is not common, because of the ease with which a discharge of gas into a room can be prevented. The preequisites of preventing CO poisoning from an oven are a good draft through an efficient chimney, and care in keeping the stove door closed. Weather conditions, such as a low barometric pressure, demand extra care in operating an oven. Most cases on record have been due usually to gross carelessness.

FIREMEN AND BOILER OPERATORS

The United States Navy Surgeons have found symptoms indicative of carbon monoxide poisoning among firemen and stokers on warships, but have

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failed to ascertain whether caused by high temperatures or CO gas. The location of boiler rooms on large sea-going vessels below the water line makes ventilation and fresh air a tedious problem. Again it might be said that CO poisoning in this line on land is usually due to negligence in operation.

MILD FORMS OF "GASSING"

Mild forms of "gassing" occur in such concerns where fuel is burnt in small quantities without an outlet to the outside air. We find this to be true of salamanders, which are used for drying the plaster and paint in new buildings. Other instances to be classified under this caption include electroplating, electrotyping, stereotyping and linotyping, machine casting of metal, can manufacturing and soldering. One case of severe "gassing" was brought to the attention of the writer, as Medical Director of the Illinois Department of Factory Inspection. A painter, upon entering the room in which he intended to work, was suddenly overcome by the fumes of a salamander. His companion made a prompt rescue, but became partially affected by the gas, producing headache for several days, nausea and malaise.

MINING

CO poisoning, severe and often fatal, is found in mines after accidental explosions, since the men cannot escape rapidly enough from the poison-laden atmosphere. Characteristic of mine CO is that it is usually inodorous. It occurs most frequently in the after-damp, the gas formed by the explosion of methane, if the latter exceeds 9.5 per cent. The CO in mines not only causes "gassing," but often also serious explosions resulting in burns and injury to the body.

FROM THE EXHAUST OF GASOLINE ENGINES

With the growth of the gasoline engine for motive power, as in the automobile and the gasoline launch, has arisen another source of industrial CO poisoning. J. N. Schumacher, chemist for the city of Chicago, upon making a series of gas analyses of gas collected from the exhaust pipes of motor cars in collaboration with the writer, has obtained the information that an average composition of exhaust gas is as follows:

	Per cent.
CO_2	6.7
CO	9.3
O	1.4
Illuminants	0.3
N	82.2
H	0.0

These analyses were not made from the first sample in the morning, but after the motor was warmed, which condition is even less productive of CO gas than when the motor is cold. Upon investigation the writer found that

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headaches, dyspnœa, attacks of vertigo, nausea and other symptoms were common in chauffeurs of large taxicab garages. The chauffers unaccustomed to continuous doses of the exhaust gas became more readily affected than the machinists and others, who were constantly in the garages. The physician for one of the larger concerns informed me that this condition is more prevalent during the cold weather season.

One morning the writer's chauffeur observed that his companion, who had been standing near the back of the car while the engine was running, staggered about the garage, whose windows and door were closed. The chauffeur opened the door and resuscitated his companion. Although not in coma, stupor was present for 2 to 3 minutes. The victim said that he noticed first a pressure in the temples, then a weakness in the legs, and blurring before the eyes. Cases of death have occurred in private garages, where the occupant being alone was unable to escape from the CO gas.

Pathology.—The post-mortem findings will depend on the length of time elapsing between death and autopsy, and also on the suddenness of death. Following a very quick and rapid death, the cadaver may be pallid and cyanotic, with swollen face and neck, similar to death from CO_2 gas. Victims of CO gas at other times present a life-like picture, sometimes with rosy cheeks, and a calm, undisturbed face, as if there had been no death struggle.

Red blotches are seen on the face and body, more frequently on the former. Such findings at once suggest a spectroscopic or Haldane examination of the blood for CO. These blotches are hyperæmias of the skin, having occurred with vasomotor paralysis, though sometimes they are due to hemorrhages into the tissue.

It has been said that the odor of gas can be detected from the mouth of the corpse. If this does occur, death must have been recent, and the gas must have had other constituents, as so to impart an odor. This may be true of illuminating gas poisoning.

Where death results from CO poisoning the body is usually remarkably well preserved and retains its heat for an unusually long time. In a mine explosion at Whitehaven,[6] England, 136 were killed and found 5 months later in a well-preserved condition. The spectroscopic characteristic of CO hemoglobin was obtained in the blood of 36 of the victims.

In the interior of the body, attention will be attracted to the bright, cherry-red color of the blood which flows freely. This peculiar color of the blood is the most characteristic post-mortem sign of CO death. This blood will keep in excellent condition for months, for purposes of examination, if placed in a well-stoppered bottle. Upon examining the blood by means of a spectroscope, the two bands between D and E will be seen. How long a time after death a determination can be made has never been accurately determined, since the findings depend on various factors. In the above-quoted Whitehaven disaster it was positively proven that the spectroscopic bands can be seen 5 months after death. The conditions underground in this mine

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may have been more favorable for preservation than if death had occurred above ground.

CO often causes a rapid fatty degeneration. This may be observed in the heart muscle, liver, kidneys, vessel walls, and other tissues.

Another change is the occurrence of small hemorrhages, usually in the brain, though also in the muscles, lungs, and other tissues.

The heart upon being opened shows bright red blood, sometimes fatty degeneration in the musculature and in the walls of the large vessels.

Broncho-pneumonia is common in those having died from the effects of CO 1 to 2 weeks after the "gassing," the pneumonia being a complication. Upon cutting the lungs, the characteristic color of the blood can be observed.

The gastric and intestinal mucosa may show small punctiform hemorrhages.

Kidneys: fatty degeneration, and necrosis in the convoluted[7] tubules.

Skin: herpes, blebs, pemphigus and gangrene.

Carbon monoxide frequently causes pathological changes in the brain. Changes have been observed in those who have died from the immediate effects, and among those who have died from psychoses. Cerebral hemorrhages and thromboses in cerebral vessels are not uncommon. The hemorrhage may be extensive or there may be numerous punctiform hemorrhages. Mott[8] records in one post-mortem that he found various congestive patches about the external and mesial surfaces of the hemispheres, with all indications of subpial hemorrhages. Autopsy on cases from psychoses of CO gas shows a predilection for degenerations, thrombosis, encephalitis[9] in the lenticular nucleus and the optic thalamus. It is held that the encephalitis and hemorrhages sometimes observed in gas autopsies are due to the rapid fatty degeneration, which occurs in the vessel walls. Kolisko[33] states that symmetrical softening in the brain, especially the lenticular nucleus, is such a common finding in those having died of illuminating gas that it is of great medico-legal importance.

Another common finding in post-mortems upon "gas" cases is extensive hemorrhage into the serous tissues, pleura and peritoneum.

In summary we find that:

1. Carbon monoxide produces a characteristic blood finding, both in gross appearance and as seen by the spectroscope.

2. That pathological changes in the tissue are not constant.

3. That broncho-pneumonia is a frequent pathological complication.

4. That CO causes fatty degeneration in the blood-vessels and other tissues, and extensive hemorrhage into the serous tissues.

5. That CO has a predilection for causing degenerations in the brain, also thrombosis, encephalitis, and hemorrhages.

Medico-legal Question.—Can carbon monoxide enter the body after death?

The interesting question has often been raised whether the detection of

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carbon monoxide in the blood is unmistakable evidence that poisoning by the gas had taken place, or whether death resulted from other causes, and that, by reason of the corpse having been placed in an atmosphere containing carbon monoxide, permitting a sufficient quantity of the gas to be absorbed, so as to cloud the diagnosis of suicidal or accidental poisoning or death from some other cause. Whenever carbon monoxide is found in the blood the general supposition is that it was inhaled into the lungs during life and entered the circulation in that manner. In 1902 experiments by Wachholz and Lenberger,[10] who placed the bodies of stillborn children in an atmosphere of pure carbon monoxide gas, showed that within a half hour the deep livid patches observed in the skin of the cadavera had turned to a red-rose color. The presence of carbon monoxide in the blood was determined by the spectroscope, even as late as 7 days after the immersing process, especially in the blood removed from the heart. In permitting a long period of time to elapse between death and the placing of the cadaver in the gas the chances of absorbing carbon monoxide lessen. Some German authorities, as Strassman and Schultz, venture the opinion that carbon monoxide enters the skin and external coverings of a dead person and penetrates into the blood, and that the quantity of carbon monoxide in the superficial and more exposed blood and tissues is always greater than that found in the deeper and more central parts.

When carbon monoxide is present in the blood of a dead person it is of utmost importance to determine at what time the gas penetrated—whether during life or after death. In the post-morten diffusion of the gas there is a well-defined difference between the anterior portion of the liver, whose carbon monoxide contents is plentiful, and the posterior portion, which contains less CO and is of a deeper color. Statements have been made that this difference is not noticeable when death has resulted from poisoning. In this instance the blood at the periphery contains more carbon monoxide than the deeper parts of the body. Experiments by Strassman and Schultz upon the bodies of old men offer evidence that there was no part of the body, practically speaking, into which carbon monoxide did not penetrate, depending upon the length of exposure to the gas. These experimenters, too, mention the difference between the anterior and posterior portions of the liver, as stated above, also that in cutting through the tissues the blood nearest the skin always contained the greater amount of carbon monoxide. In a case of poisoning by inhalation of carbon monoxide such a difference would not occur, since the blood would be equally affected all through the body. Failure[10] to state the quantity of carbon monoxide recovered in the blood greatly weakens the argument of Strassman and Schultz. The point in this statement will be clearer when attention is drawn to the fact that very minute quantities of carbon monoxide may be present in the blood in normal conditions. The possibility of the diffusion of carbon monoxide into the blood and tissues of a corpse is a matter of great importance from a medico-legal point of view, as the following case illustrates.

Oliver[10] reports a German case of a woman who was found dead in her

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room, with one end of an India-rubber pipe in her mouth and the other attached to a gas pipe, the valve of which was open. The room smelt strongly of coal gas. Everything seemed to favor the theory that death was suicidal, but at the last moment the father of the dead person intervened and asked for a delay in the interment as he believed an operation for the induction of abortion had taken place at the instigation of the husband, and that in order to raise the question of suicide the tube had, in order to mislead, been placed in the woman's mouth. An inquiry was ordered, and Strassman and Schultz found that no abortion had taken place, and that there were all the typical signs of poisoning of carbon monoxide.

In summary, the writer believes that, if a body is examined and it is found that all the organs and all parts of the organs present the characteristic carbon monoxide pathologic findings, especially in the liver, and that if the CO saturation of the blood is in the neighborhood of 75 to 83 per cent. as determined by the Haldane method, death was due to carbon monoxide poisoning; whereas, such cases as present a typical and a symmetrical carbon monoxide pathological finding and where the hemoglobin saturation is low, reasonable doubt may there exist as to the cause of death.

Symptomatology.—An acute carbon monoxide poisoning will vary in intensity upon the amount of CO which enters the blood, and upon the rapidity with which it is accomplished. Other determining factors are the susceptibility of the person "gassed" and whether pathological accidents such as hemorrhage occur with the initial insult. Among four workmen who seemed exposed to equal quantities of the gas, the writer observed that one was in deep coma, whereas the others were simply in stupor.

Prodromata.—In some cases of "gassing" the victims succumb as rapidly as if struck by lightning, with no apprehension of its presence and with no prodromes. The coma and even death may be almost immediate. Out of about 300 cases of industrial gas poisoning comas observed the writer would say that about one-third occurred without prodromes. Absence of prodromes is especially true of blast-furnace gas intoxication.

Following are the more common prodromes of carbon monoxide poisoning: a throbbing or feeling of pressure in the temples; tinnitus aurium; distress in the epigastrium; nausea, sometimes vomiting; weakness in the legs together with sluggishness of movement; hallucinations and wild mental pictures; sometimes convulsions; a severe or even bursting headache; and blurring before the eyes.

The most common prodromes are: the pressure and throbbing in the temples; the epigastric pain; and the weakness in the lower limbs. The latter is often described as a "caving in" at the knees and an inability of movement. Some "gases" are able to move away and escape, others seem "glued" to the spot, and are unable to conduct locomotion. From the prodromes, if they occur, the patient lapses into somnolence, then into coma.

Dr. C. Le Neve Foster[3] makes some interesting statements concerning the

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sensations and symptoms experienced upon being "gassed" in mine air containing about 1.07 per cent. CO. "The poison took effect most suddenly. . . . I felt decidedly queer, when I reached the level and thought that a drop of brandy might revive me. I took out my little brandy flask, but already my fingers seemed incapable of doing the work properly. . . . Everything seemed in a whirl, and the atmosphere seemed to be a dense white fog. . . . A curious fact is that we all sat without moving or trying to escape; the foot of the ladder was close by, yet none of us made any effort to get to it and even ascend a single rung." In writing a note of farewell to his family he states: "I frequently wrote the same sentence over and over again." This reiteration of words and syllables is occasionally observed among the early symptoms of gas poisoning.

Other prodromes sometimes observed are: a dryness of the throat, backache; heart fluttering or palpitation; and sensations of chilliness.

CARBON MONOXIDE COMA

When the narcotic action of the gas continues, the victim becomes comatose. The usual text-book pictures of gas coma do not always occur. We are taught to look for rosy cheeks and bright red lips. On the other hand, cyanosis, palor and distorted facial expressions are frequently met with. Nevertheless, in a great number of cases red blotches and areas of hyperæmia form upon the skin and the lips and conjunctiva are a bright red. The writer has observed that these typical gas comas are more often found in illuminating and coal-gas poisoning than in those which occur from the other industrial gases.

The coma in the average industrial "gassing" is milder than those observed by physicians in the average illuminating gas poisoning where such has occurred accidentally or with suicidal intent. That a far lesser number of "gassings" accompanied with coma take place may be gathered from the fact that out of 281 "gassings" about a blast furnace only 65 were actually comatose, whereas 216 never entertained more than a stupor or the prodromal symptoms.

The reddened areas or blotches are due to vasomotor disturbances causing local hyperæmias and sometimes from actual hemorrhage into the skin. They are most commonly observed on the face.

The pulse is at first full and slow with a rise of blood pressure. Later as the coma deepens or as the patient survives, the pulse becomes feeble and rapid. Rapidity of pulse or tachycardia will continue for several days after recovery from the coma. The respirations are at first deep, stertorous, later weak and rapid.

Hyperpyrexia occurs most commonly, the temperature ranging from 100° F. to 104° and 105°. Temperature is often an indication of pneumonia. If the rise is sudden after the patient has recovered from the coma, or if it

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occurs during the coma it is suggestive of brain insult such as hemorrhage, or thrombosis. Dr. W. Gilman Thompson[23] states that in nearly all comatose cases observed by him there was an elevation of body temperature, lasting from a day to a week or more. In some cases there was a preliminary fall to 96° or 97°F. The writer observed that similarly to fatal cases of brain injury, such as cerebral hemorrhage, sunstroke and skull fracture, there is in fatal cases of gas comas an antimortal rise of temperature in many cases from 107° to 110°F.

The odor of gas can sometimes be detected on the breath early in the coma. This occurs more often in illuminating gas intoxication than with those "gassed" industrially. The odor is not that of the gas, but rather of a characteristic odor familiar to the physician meeting frequently with gas comas.

Nervous phenomena observed are initial convulsions, twitchings of muscles during the coma, and absence of the deep reflexes (depending on the depth of the coma). Rare signs are nystagmus and strabismus.

Other incidents of gas coma are involuntaries, Cheyne Stokes respirations, local œdema, hemorrhages in the sclera and conjunctiva.

No diagnostic aid is given in a urine analysis. Albuminuria is of no value nor is melituria. Four out of five successive "gassing" cases reported by a German clinician have had a glycosuria. Dr. Herbert S. Carter, who made urine analyses for Dr. W. Gilman Thompson[23] in five non-fatal cases, found an increase of chlorides and urea.

The duration of a coma varies from a few minutes to several days. Comas lasting over 48 hours are usually fatal, either directly or from a complicating pneumonia. Of course this is the probability, not the rule.

A positive diagnosis of gas cannot be made from any of the above-mentioned symptoms and physical signs. The clinical observations of a comatose patient may disclose a bright redness of the blood, cherry red lips, red blotches on the skin, and the odor of gas on the breath. Still, the most determining factor in the diagnosis is the history and especially the examination of the blood.

Analysis of the Blood.—Carbon monoxide in the blood may be detected by (1) a spectroscopic examination, (2) Haldane's method, and (3) various other color determinations.

Spectroscopic Examination.—The spectroscopic examination of the blood will positively tell whether CO is in combination with the hemoblogin of the red blood corpuscle. Hoppe-Seyler first described the carboxyhemoglobin picture in 1865. Upon placing a sample of CO blood in a small glass tube in the spectroscope, two absorption bands are seen in that portion of the spectrum described as D-E. These bands which are found between D and E have nearly the same width and are of the same intensity. They are similar to the bands of oxyhemoglobin, the band nearer to D almost coinciding, the other being nearer the violet end of the spectrum than in oxy-

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hemoglobin. The distance between the bands of carboxyhemoglobin is also slightly wider than the distance between the two bands of oxyhemoglobin.

When the blood has a saturation of 27 per cent. CO, the addition of a blood-reducing agent, as a drop or two of a solution of crystallized ammonium sulphide, does not convert or reduce the carboxyhemoglobin to one of reduced hemoglobin. This differentiates carboxyhemoglobin from oxyhemoglobin. The reduced hemoglobin has only one band in the D-E space.

Blood exposed to the air for a week often loses its CO spectroscopic picture, whereas blood sealed in a glass tube remains undisturbed for a long time for purposes of examination.

In a non-fatal case of carbon monoxide poisoning the characteristic spectroscopic blood cannot be seen after several days. Kobert[5] mentions the report by Wacholz of carbon monoxide in the blood 7 days after exposure to the gas; Fischbein reports one of 5 day's duration. In the cadaver the CO may persist for spectroscopic examination for a much longer time, as in the Whitehaven disaster where it was observed 5 months after death had occurred.

The Haldane Method.—The principle of this method is color-metric comparison.

A few cubic centimeters of normal blood solution are shaken to saturation with a like volume of air which then is ready to be tested for CO. A known volume of normal blood solution is then taken and standardized and carmine added from a burette until:

1. The quality of pinkness is reached, similar to the solution saturated with the air sample; and,

2. Equality is reached for the same blood solution fully saturated with CO is reached.

From the two results the percentage saturation of the blood solution with the sample of air is deduced; and from this the percentage of CO in the air sample can be calculated. This method is well adapted to samples of air or other gases containing 0.2 or less of CO, showing its degree of delicacy.

This test can be used both to analyze a sample of air for the presence of CO and also the blood for its CO saturation.

Other Chemical Tests.[1]—Blood rich in CO when boiled yields a brick-red mass; ordinary blood becomes brown (Hoppe-Seyler).

Ten cc. of a 2 per cent. solution of blood with carboxyhemoglobin will yield a bright red color, if 2 cc. of yellow ammonium sulphide and 30 per cent. acetic acid are added; normal blood under the same reagents gives a green precipitate (Katagama).

If a carboxyhemoglobin blood is added to diluted hydrogen sulphide water, drop by drop, a bright red color is produced; with normal blood a bright green (Salkowski).

Normal blood when shaken with one or two volumes of a solution of sodium hydroxide of 1.3 specific gravity, becomes black, in layers greenish brown; CO blood remains red like red lead (Hoppe-Seyler).

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A mixture of calcium chloride and sodium hydroxide gives a carmine-red color, if CO is present, whereas normal blood becomes brown (Eulenberg).

Equal quantities of blood (2 cc.) and water plus 3 drops of a one-third saturated solution of copper sulphate give in CO blood a brick-red precipitate, whereas normal blood has a greenish-brown color (Zellski).

Four or five volumes of lead acetate solution are added to one volume of carbon monoxide blood and thoroughly shaken for about a minute. Such blood retains its red color; normal blood under similar conditions becomes a bright red (Rubner's test).

Fischbein[34] states that the test suggested by Wetzel, namely, dilution of the blood, one part to four with water, and shaking with three times the volume of 1 per cent. tannic acid, whereby normal blood becomes gray and carbon monoxide blood remains red, is the most reliable and trustworthy test in the presence of formaldehyde (embalmed bodies). In case no formaldehyde is present Rubner's test yields satisfactory results, except that it does not act immediately but is best interpreted after several days.

Late Symptoms.—When a person wakes from gas stupor or coma, it usually takes from 2 to 3 days, and sometimes longer, for restoration to a normal condition.

The coma leaves the patient with a severe headache which lasts the entire day or persists for one or more days. This is even true of mild industrial "gassings." Moreover, anorexia, sometimes nausea, pains in the limbs and even neuritis follow a "gassing."

Twenty-seven men were critically observed for 1 week following a blast-furnace intoxication. One man who had been hardly "gassed" suffered a coarse tremor, anorexia and insomnia for 3 days. Tremor of the tongue and fingers is a common aftermath. One of the above-mentioned 27 was markedly neurasthenic for 1 week, and also complained of anorexia and insomnia. Even severer after-effects have been observed by other physicians.

Sequellæ.—Among the sequellæ of carbon monoxide poisoning are: (1) pneumonia, (2) psychoses, (3) paralyses, (4) skin eruptions, and (5) gangrene.

Pneumonia.—Broncho-pneumonia frequently terminates a gas poisoning. It occurs both during the coma and often when the patient seems entirely recovered. Thus we find pneumonia following a CO intoxication lasting from 2 days to 2 weeks. Conditions which favor its occurrence are the length of the coma, carious teeth, exposure to cold, and the respiratory irritation of the gas and of oxygen used in resuscitation. The pneumonia is commonly of the "schluck-pneumonie" type. The inordinate use of oxygen has been thought by one of the physicians of a large illuminating gas company to be a respiratory irritant and, therefore, a predisposing cause to pneumonia. Perhaps it may be that the oxygen was inordinately used in such cases where the coma was deep, as pneumonia might have occurred also without using oxygen.

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The Psychoses.—Psychoses must not be interpreted to mean those milder phenomena which follow "gassing" immediately, such as neurasthenia, muscular twitchings, insomnia and other mild neuroses. Definite mental disorders have been reported from various sources in the literature following carbon monoxide poisoning. Sibelius[13] and Stierlin[11] have presented this phase of the subject most thoroughly and in admirable detail. Others who have treated of carbon monoxide psychoses are Zangger,[14] Giese,[18] Mott,[8] Libin,[9] Weidner,[15] O'Malley,[16] and Abrahamson.[11] Sibelius classifies this complication as (1) psychoses directly due to CO, (2) psychoneuroses, (3) emotional psychoses, (4) traumatic or mixed psychoses. The most concise understanding of the subject for the industrial hygienist is the knowledge that some CO psychoses follow "gassing" immediately and that others have a lucid interval of from 2 to 3 weeks, keeping in mind that actual brain lesions may occur due to CO, causing thrombosis and degenerations, while still others might be traumatic, precipitating the psychosis where the victim is neuropathically predisposed.

How commonly do we find psychoses following industrial "gassing?" The literature, rich in descriptions of psychoses, fails to show any attempt at separating those cases which occur industrially from those chargeable to neurotic individuals with suicidal tendencies. The writer believes that if an investigation were made, few cases of psychoses following industrial "gassing" would be discovered. Dr. James Burry, Surgeon in Chief of the Illinois Steel Company, in his extensive experience with "gassing" has never observed one single case of psychosis undisputably attributable to carbon monoxide.

In comparing the reported psychoses subsequent to carbon monoxide poisoning, no two cases present the identical clinical picture. We find that they simulate dementia præcox, acute mania, general paresis, Korsakow's syndrome and other forms of dementia. O'Malley's case had amnesia, apathy, stupor, delirium and confabulation. Giese[18] reports a case having amnesia, stupor, and a resemblance to Korsakow's syndrome, terminating fatally. Dr. Alex Scott quotes a case of dementia with acute mania, of 8 days' duration, with recovery. Dr. Sanger Brown, of Chicago, also reports a case with both retrograde and anterograde amnesia following a "gassing." The patient never recovered entirely and died suddenly 8 months after exposure to the gas.

At this point the writer quotes a psychosis which occurred to a young man who was "gassed" while working above the bustle pipe of a blast furnace, where he was found in deep coma, after having fallen for a short distance. He remained in coma 12 hours in the company's hospital. Subsequently he became delirious, restless, unable to deintify relatives and friends. He suffered from involuntaries, muscular twitchings and apathy. He could not realize what had happened to him. When the delirium ceased, he remained very stuporous for several weeks in the hospital. Above in-

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formation was obtained from relatives and hospital physicians. When first examined by the writer, this patient had been home for 4 months. During this time he had tried to work, but was unable to find his way about the company's plant and grounds.

Following facts were obtained: The patient had previously been an industrious person. He used whiskey and beer moderately, was married, had one child at the time of the accident. He lived alone, having previously acquired syphilis from his wife. A Wassermann test was medium positive. At the time of examination he suffered from an amnesia which was characterized by a loss of memory for all events except those which occurred in his childhood. He remembered Dr. Karasek and the writer at intervals of several days, but in many other instances his memory lapsed even on daily happenings. Thus he had a retrograde and partial anterograde amnesia. He could not orient himself on the streets and became lost even though a few doors from his home.

His reasoning power did not seem affected. He understood that he was sick, was able to talk rationally, and identify many objects.

His speech was slow and similar to a paretic. His gait was slow and awkward. The dynometer showed diminished muscular strength. Reflexes were normal, the patellar being sluggish.

Although this case showed a positive Wassermann, the patient, as compared with other gas psychoses recorded, evinces a great similarity. He had an amnesia, loss of orientation, and other signs, which appeared suddenly upon being "gassed." Moreover, he had been in good health before the accident.

In conclusion, psychoses upon carbon monoxide poisoning are characterized:

1. That they occur either directly after or at a shorter or longer interval after the "gassing."

2. That they are variable in type, often simulating other forms of psychoses, such as dementia præcox, general paresis, and Korsakow's syndrome.

3. That amnesia or a loss of memory is the most common finding in gas psychoses.

4. That the pathological basis is brain hemorrhages and thrombotic occlusions, with encephalitis as the result of carbon monoxide poisoning.

5. That the majority recover, and that no prognosis can be divided from the character of the psychosis.

Cerebral Hemorrhage.—Cerebral hemorrhage with hemiplegia is sometimes the result of an acute carbon monoxide poisoning, due to the rapid fatty degeneration of the blood-vessel walls, also to the rise of blood pressure at the onset of the coma. Such hemorrhages also cause other forms of paralyses or motor disturbances. The English Factory Inspector reports the case of a fatal cerebral hemorrhage in a man 24 years old, who while cleaning a main

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was "gassed." In connection with the paralyses occur disturbances of sensation, as paræsthesias, analgesias, and hyperæsthesias.

Neuritis.—Actual neuritis is not as commonly seen as with other occupational poisons, such as from lead, mercury, and arsenic. Nevertheless, we find neuritis reported in the literature. Lancereaux mentions a paralysis of the leg due to a carbon monoxide neuritis. Krantz[19] refers to a polyneuritis following gas poisoning.

Other nervous complications to be enumerated are deafness, amblyopia, and diplopia. The English Factory Inspector reports a case of diplopia lasting 22 days following a carbon monoxide "gassing."

Gangrene.—The writer has never personally seen this complication, but refers to those reported by Alberti[20] and McLean.[21] In the former's case there appeared soon after "gassing" blebs containing a brownish fluid material, showing a carbon monoxide spectroscopic picture. Later extensive gangrene of the elbow, lower limbs and the neck, with a lethal termination, developed. In McLean's case there were scattered areas of gangrene in the legs. The pathological basis of gangrene with carbon monoxide poisoning is uncertain, some considering it due to thrombosis, others to trophic and degenerative changes in the tissue itself.

TREATMENT OF GAS POISONING

Prophylaxis.—In this chapter the writer will deal only with the prevention of acute industrial "gassing."

The prevention of industrial "gassing" is dependent on (1) the employer, (2) the employee, and (3) the state.

The employer is responsible for the placing and operating of gas-producing machinery, and the administration of safety rules for his employees.

The large steel companies have been on the alert to adopt every modern appliance with a tendency of preventing gas poisoning. The Illinois Steel Company's safety department, composed of both employers and employees, continually considers the methods and means of preventing "gassing." The following methods have decreased gas poisoning in steel mills to a minimum in the opinion of the writer. First, the adoption of automatic top-filling furnaces (see pictures comparing old and new style furnaces); second, the use of overhead mains instead of underground; third, reduction of leakage by improved methods of lining and riveting the furnace and pipe walls; fourth, by enforcing adequate safety rules.

The illuminating gas companies and large producer gas users must also be commended in Illinois for improved methods of prevention.

The employee also shares responsibility in his work of prevention. He must learn to recognize the danger of gas and its sources. Of dangerous occupations it might be said that familiarity breeds contempt. Reference

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is here made to the switchman, who, in spite of persistent warnings, will jump the cowcatcher of the engine while in motion. Alcohol should be abstained from during or before working hours. Some men have gone to sleep under the influence of alcohol alongside the comfortable warmth of a furnace to be found by his fellow-workers fatally "gassed."

The state is advancing in factory regulation. Nearly all civilized governments have adopted laws for the prevention of industrial accidents and diseases. The state of Illinois does not control gas poisoning by its Occupational Disease Law, which provides more specifically for disease from lead, brass, zinc, arsenic, and phosphorus, but regulates the danger from gas poisoning by means of the Health Safety and Comfort Law, which provides for safe machinery, necessary ventilation and the safe removal of obnoxious and poisonous dusts and gases.

The following laws are enforced by British Factory Inspectors:

1. Notices are exhibited at different parts of the works calling attention to the dangers of the gas.

2. Men engaged in flue cleaning are never permitted to work single handed nor are both men permitted to enter the tube at the same time. They can watch their mate by turn.

3. No workman is allowed to enter the tube until it is cleared of gas. External work, such as raking out the dust from the bottom doors, is always completed first.

4. During cleaning, a cylinder of compressed oxygen, connected to a pulmotor, is kept on the spot. Various persons have been instructed in the use of the pulmotor, which is a mechanical device for causing artificial respiration at the same time supplying a 60 per cent. mixture of oxygen and air.

5. Attendants are supplied with ropes for rescue purposes, and a smoke helmet with bellows and piping, which the attendants would use in order to enter the tube to tie on the rope to any one "gassed," and assist him out of the tube.

Following are regulations adopted by the Illinois Steel Company pertaining to "gassing:"

1. "Never go into a gas-engine house or electric motor room unless your duties take you there, and you have special instructions from your foreman.

2. "Work around blast furnaces is very dangerous, because of escaping gas, the possible breaking out of the side of a furnace, explosions, and the blowing of hot stock out of the top of the furnace. These dangers cannot always be guarded against by those in charge, and every one near a furnace must be watchful for his own safety.

3. *Gas May Kill You.—Dangerous* gas may be found anywhere. Sometimes you cannot see or smell it. It may make you *unconscious* before you know gas is present. Sometimes gas makes you feel dizzy, or gives you a headache, or stiff neck or weak legs. When you feel any of these symptoms, get into fresh air.

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4. "Employees are forbidden to go top of a furnace without the permission of the General Furnace Foreman.

5. "Before working around the explosion doors, bell rods or hangers, or doing any work between the big bell and gas seal, see that the gas valves are closed, that the gas escaping from the stock is lighted and that the man operating the bells is notified.

6. "Before changing tuyères, notice should be given the party in charge of filling the furnace to stop filling until he gets notice that tuyères have been changed. This is to prevent gas from blowing out on the men through the tuyère openings of the furnace.

7. "Keep away from valves under dust catchers and down legs, especially when a furnace slips. There is danger from gas.

8. "You must not go anywhere above the floor, around a blast furnace (around bustle pipes, water troughs, valves, roofs, stoves, etc.), without first telling the General Foreman on the Blower of that furnace.

9. "Stove Cleaner Foreman must order men out of stoves when the wind is off the furnace or when a furnace is slipping badly, as there is danger of gas from the Furnace backing into the stoves and igniting.

10. "Keep out of the engine rooms unless your duties take you there. There is great danger from gas.

11. "Never work in the basement alone—always have a helper with you. *Gas may kill you.*

12. "If you find a man 'gassed,' get him into the open air and call foreman at once.

13. "Gas may be found anywhere; never go into holes or out of the way places alone."

Resuscitation.—The above-mentioned regulations show clearly how the workman is removed from a gas atmosphere without "gassing" more men. Resuscitation is then begun. This is usually conducted at the site of the "gassing" up to the point where the patient can be safely removed to a hospital or his home. Many industrial "gassings" do not demand much more treatment than removal to pure air. Not all are deep comas; in fact the majority are mild in which the patient is only in a simple stupor.

Artificial respiration should be begun as soon as possible, either by attendants or by means of a pulmotor. Tanks of oxygen and pulmotors are kept close to the furnaces in many companies. Experimental work performed by Haldane shows that an atmosphere of oxygen keeps the patient alive much longer than an atmosphere of air. The writer does not deem it necessary to go into detail as to the relative merits of using oxygen in pure state or the pulmotor with a mixture of nitrogen and oxygen. Either may be used for resuscitation, but great care should be employed.

The patient should be wrapped in blankets or be removed to a dry, warm place.

After removal to a hospital the writer recommends, if the pulse is strong

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and the blood pressure not subnormal, a venesection of 500 cc. of blood with the simultaneous administration of normal salt solution by hypodermoclysis.

Stimulants such as caffeine, strychnine, and camphorated oil are also useful. Catharsis and sedatives are of use for the distress which for a few days follows a gas intoxication.

Sequellæ.—Time does not permit a discussion of the treatment of the sequellæ. Moreover they come properly under the direction of the internist, surgeon, and psychiatrist.

CHRONIC CARBON MONOXIDE POISONING

Sommerfeld[22] states that chronic carbon monoxide poisoning produces headaches, vertigo, nausea, vomiting, coated tongue, amnesia, anæmia, flushes, formication, heart palpitation, nervousness, and malaise. It is true that many persons who are continually subjected to small continuous doses of the gas are sometimes afflicted with the symptoms mentioned above. The writer has personally seen undisputed cases of chronic monoxide intoxication, confirming such by means of a method which will be mentioned later. Nevertheless, upon critical examination of 240 men, in 1910, together with Dr. Matthew Karasek, and upon further investigation since then, the writer must conclude that a worker can acclimate himself to CO within certain limits. An explanation will be offered for this in a subsequent chapter.

Carbon monoxide is found in small quantities in the steel companies about the blast furnaces, about producer furnaces, illuminating gas plants, about the street gas mains, in motor garages, in rooms where electrotyping, stereotyping and monotyping is done, in foundries, above the machine solderers, in can making, in smelters of ores, in rooms where salamanders are being used, in laundries and bakeries, and in tailor shops from the ironing goose.

In making quantitative analyses on air collected in such establishments as are mentioned in the previous paragraph, the writer has found a great variation in the CO present, at different times and in different parts of the same room. Analyses have been made in motor garages, above the soldering kettle of a linotype machine, and in tailor shops while the goose was being used, in collaboration with J. N. Schumacher.

In five analyses made in motor garages the CO content of the air was as follows:

	Per cent.
1. CO	0.02
2. CO	0.13
3. CO	0.04
4. CO	0.00
5. CO	0.02

The average for above five samples was 0.042 per cent. CO.

We recommend the following method for obtaining qualitative and quantitative analyses of CO in suspected rooms.

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A glass sampler, such as is ordinarily used for collecting gases, is completely filled with water, and simply allowed to drain in the atmosphere of which a sample is desired. The exit of water causes the entrance of the desired air or gas. The sample is now ready for analyses, having only to be displaced into any apparatus, which may be selected for analysis, depending on the analytical process selected. Using the pentoxide of iodine method, for instance, the gas is caused to pass through a tube containing KOH, which removes CO_2, then through H_2SO_4, which in turn eliminates any alkali and moisture. From here the gas passes through the pentoxide of iodine which is contained in a tube surrounded by a heated oil bath, 300°F. It is here that the carbon monoxide reacts with the pentoxide of iodine so as to set free iodine, which is then absorbed in a solution of KI (0.5 gram to 10 cc. of water) and subsequently estimated quantitatively by titration.

The qualitative determination simply demands a starch solution into which the CO liberates the iodine, turning the starch solution blue.

The method* of titrating the KI containing the I liberated by CO is to titrate this solution against thio-sulphate N/1000.

Pathology.—The pathology of chronic carbon monoxide poisoning does not refer to post-mortem findings, since there exists no cause of death from chronic intoxications, but to the clinico-pathological examination of the blood.

During the Illinois investigation of 1910, a critical examination was made of 240 men who had either recently been "gassed" or who had been subject to frequent "gassings." During this work the blood of 68 men was examined by Dr. Matthew Karasek and the writer, with the following observations:

1. That there existed a polycythemia in these cases of 5,500,000 to 9,676,000, 66 per cent. being over 6,000,000 cells per cu. mm.

2. No cell such as poikilocytes, normoblasts, microblasts, etc. No embryonal red cells.

3. Hemoglobin estimated from 95 per cent. to 125 per cent., more estimations being over 100 per cent. than under. Whites 7000 to 10,000.

Color index under five-sevenths.

6. Eosinophiles in about two-sevenths cases from 5.5 per cent. to 8.5 per cent., myelocytes in one-third cases as high as 3 per cent. Basophiles rare.

Similar work was done by Drs. Nasmith and Graham[32] on guinea-pigs. They allowed the animals a saturation of 25 per cent. of the coloring matter of the blood, with carbon monoxide. The guinea-pigs inhaled this gas daily for several weeks. Although their blood was saturated with 25 per cent. CO, they did not loose weight, become anæmic, nor show any signs of disturbance from the gas.

Following is quoted from the work of Drs. Nasmith and Graham: "With 25 per cent. of its blood rendered useless for oxygen-carrying

* $2NaS_2O_3 + 2I = 2NaI + Na_2S_4O_6$.

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purposes, by its union with CO, the guinea-pig is capable of compensating and will manufacture new red blood corpuscles until it has reached a total of 8,000,000 with a corresponding hemoglobin content of 105 per cent. The normal number of erythrocytes in the peripheral circulation of guinea-pigs is about 6,000,000 with 88 per cent. hemoglobin. The animal with true compensation has three-fourths of 8,000,000 or 6,000,000 erythrocytes and three-fourths of 105 per cent. or 79 per cent. hemoglobin still available. Thus there is only a total loss for oxygen-carrying purposes of 9 per cent. of its hemoglobin."

Following were their conclusions of their entire experiment with CO:

1. "CO acts as a poison solely by its ability to prevent the normal supply of oxygen from reaching the tissues, and thereby deranging the normal metabolism of the body cells.

2. "Guinea-pigs living continuously in a dilute atmosphere of carbon monoxide, so that the oxygen-carrying power of the blood is reduced, are able by increasing the quantity of hemoglobin and number of erythrocytes to compensate for this loss and maintain an oxygen-carrying capacity approximately equivalent to that of the original blood.

3. "CO poisoning is followed by a leucocytosis of the eosinophile and pseudo-eosinophile forms. A moderate saturation produces a moderate toxæmia involving an eosinophilia like nearly all moderate toxæmias. A high saturation causes a severe toxæmia in the course of which the eosinophiles disappear, as in all severe toxæmias. A high prolonged saturtation brings about the appearance of erythroblasts and myelocytes, indicating hyperactivity on the part of the parent cells in the bone marrow.

4. "The effect of CO in increasing the number of the erythrocytes in the blood is, in many respects, similar to those of high altitudes, in the peripheral circulation at least."

The writer at this point presents a partial representation of work done on acute and chronic "gassing" in the period from 1910 to 1914.

TABLE I

Case	Duration of coma	Red count	Red count third day	Red count sixth day	HB (Dare)			White count
					1	2	3	
					%	%	%	
Female, illuminating gas	10 hours	4,876,240	5,200,400	4,906,430	80	90	90	19,460
Male, 42 years, illuminating gas	23 hours	5,267,000	5,640,300	5,602,100	90	100	100	18,760
Male, 47 years, illuminating gas	3 hours	5,100,800	5,706,060	5,980,760	95	95	95	16,600
Male, 31 years, blast-furnace gas	6 hours	4,878,410	5,146,700	5,317,000	95	100	100	12,860
Male, 39 years, illuminating gas	27 hours	5,163,700	6,821,300	6,251,100	95	100	100	9,791

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TABLE II

Sex	Occupation	How long employed	Red blood count	Symptoms noted
Female	Tailor shop	6 years	5,762,100	Headaches
Female	Laundry	1 year	6,432,130	Headaches
Male	Can manufacturer	22 years	7,611,310	Headache, anorexia
Male	Can manufacturer	10 years	6,421,140	Neurasthenia
Male	Can manufacturer	4 years	5,280,710	Neurasthenia
Male	Salamander	3 years	8,497,600	Headaches, anorexia
Male	Salamander	5 years	6,311,000	Nausea
Male	Salamander	5 years	6,131,000	Headaches
Male	Salamander	12 years	4,376,000	Headaches
Male	Tailor	5 years	6,378,000	Headaches
Male	Tailor	6 years	5,810,000	Headaches

The above-mentioned work gives reason for saying that the blood-forming organs, tending to compensate a loss of oxygen-carrying power in many individuals, cause an increase in the number of red cells and the quantity of the hemoglobin.

Symptomatology.—Since acute "gassing" may occur in so mild a manner so as to produce only a headache, it is easy to confuse acute and chronic carbon monoxide poisoning. When mention is made of certain symptoms as being so-called chronic carbon monoxide poisoning, we must eliminate the possibility of error by investigating whether any certain patient presents such symptoms continually or at regular periods, and it is also well to understand that such a person is continuously exposed to small quantities of this gas, or is "gassed" frequently in a very mild manner.

Persons who will not acquire a tolerance to the gas will become anæmic, irritable, neurasthenic, and complain of headaches, anorexia, loss of weight, backaches, vertigo and gastro-intestinal disorders.

Whether the compensating power of the blood-forming organs in those persons living with a polycythemia will ever exhaust itself, as in the case of a hypertrophied leaky heart, is yet to be determined. The writer has not been able to find a greater incidence of pernicious anæmia among blast-furnace workers.

The diagnosis of chronic carbon monoxide poisoning was recently made by the writer in the following manner. A person engaged as a mechanician in a can-making concern was referred to the writer by Dr. William E. Evans, of Chicago, to determine the occupational element in the man's disorder. It was necessary in the patient's work to occupy himself for certain periods every day over a burning machine solderer, during which time it was possible to inhale carbon monoxide fumes. The patient complained of a symptom-complex very much like that usually mentioned under chronic CO poisoning. Upon excluding tuberculosis, as far as my ability permitted me, nephritis, and especially lead poisoning, the latter by means of a blood examination

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for stippling and a urine analysis for lead, a red count was obtained in the neighborhood of 7,000,000 red cells. The diagnosis of chronic carbon monoxide was made. Upon changing his manner of work the patient obtained relief.

A loss of muscular power seems to occur in those subjected to carbon monoxide poisoning. This should be referred to as a sign of chronic intoxication. Dr. Matthew Karasek and the writer made comparative tests on the workmen about a blast furnace, and on those working in non-gas productive industries, selecting as much as possible workers of the same nationality and habits. Diminution in strength, as noted by the hand dynometer, was noted in those subject to the gas.

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SECTION V

LEAD POISONING

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Although lead poisoning has existed from time immemorial, we yet learn little or nothing of it from Egyptian history. When Rome was at the height of her power and the supply of water for drinking purposes and for baths became a pressing requirement, also when the question had been partly solved by the large aqueducts built by her engineers, the use of lead pipes for domestic distribution became more or less a necessity. That the people suffered from drinking lead contaminated water is evident from the condemnation of the metal by Vitrivius, the Roman architect. In one of the large Roman Camps near Newcastle-upon-Tyne I found lead pipes which had been used for the conveyance of water through the Camp. The Romans in making lead pipes did not mould the pipe as is done to-day, they simply beat flattened lead into a tubular shape.

In medical literature lead poisoning occurs under various names, *e.g.*, *saturnism*, *plumbism*, *colica pictonum* and *colica pictorum*. The sources of the poisoning are industrial and accidental. Occasionally lead poisoning has assumed epidemic proportions: it has broken out in a town owing to the drinking water possessing strong plumbo solvent properties. The "gathering ground" has much to do with the plumbo solvency. A peaty soil is apt to render water slightly acid, owing to humic and ulmic acids, and to confer upon it plumbo solvent properties. Rainy weather succeeding a long drought also favors the disposition of the water to attack lead. The presence of certain microörganisms in peaty water is not without considerable influence. Houston found two non-motile, non-liquefying bacteria in peat; these possessed separately the properties of acidity and plumbo solvency. Distilled water attacks fresh lead pipes with considerable vehemence. The presence of oxygen in the water favors the action; so too does carbon dioxide. On the other hand, the presence of calcium carbonate confers a protective influence so long as the water remains free from acid. Nitrates and chlorides in water tend to corrode lead. Silicates possess protective properties. The plumbo solvency of drinking water can be considerably reduced and the water rendered safe for personal and domestic purposes by the addition of fragments of limestone, magnesium limestone and chalk, to the filtering beds, but care must be taken even with those, since the addition of too large a quantity of chalk, for example, may be followed by results, the direct opposite to those which were expected. There is a limit in regard to the power for good of these alkaline earths which can only become

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known by testing the plumbo solvency of the water two or three times a year. There is a difference too in the effect due to the age of the lead pipe. New lead pipes are more susceptible to the influence of water than those which are old. J. F. Liversuge and A. W. Knapp,[1] as the result of a series of experiments found that when a new pipe containing water was closed for 17 hours about half the lead removed was dissolved in the first 4 hours and that the average amount of lead dissolved from pipes increased with time up to 44 weeks. After a year the age of the pipes gains little or no further protection. Different parts of a lead pipe yield different amounts of lead to water under similar conditions. This circumstance and that of temperature are partly responsible for the fact that the water removed from a tap in a certain part of a town may possess different plumbo solvent powers to that drawn in another part of the town. It was found that the lead pipes made in Paris responded less to the influence of water than those brought from Nantes. Water drawn from one of the large hospitals in Paris was found to contain 55 mg. of lead per liter, whereas water drawn in a house in a different part of this city, but at the same time of day, contained only 1.6 mg. of the metal. The presence of metals other than lead in the pipe induces electrolytic action which causes dissolution of lead. Where pipes containing lead, antimony and tin have been substituted for ordinary lead pipes the results have been disastrous.

It is impossible to say definitely what amount of lead in drinking water is dangerous. Much depends upon the amount of water drunk by the individual. Water containing 1 to 2 mg. of lead per liter might have little influence upon a person who consumed 1000 cc. in 24 hours but the results would be different if 2 liters or more were drunk daily. Toxicologists are of the opinion that water which contains 5 mg. of lead per liter is dangerous. It is the daily absorption of minute quantities of lead which is harmful to the organism. Since water standing in the pipes over night may have dissolved a considerable quantity of lead, the tap ought to be turned on and the water allowed to run every morning for few minutes before it is used for culinary purposes. The drinking water for the table should be passed through a carbon filter rich in animal phosphate.

Another accidental source of lead poisoning is food—especially tinned food. An acid fruit such as pineapple is particularly prone to attack the lead present in the solder of the tin. One of my worst cases of lead poisoning was that of a lady librarian. She suffered severely from colic and double wrist drop in consequence of having eaten almost daily for months tinned salmon.

Persons addicted to the habit of snuffing occasionally suffer. One of my patients was in the habit of keeping the snuff in lead foil. Discovery of the cause soon led to disappearance of the symptoms.

Industrial lead poisoning is extremely prevalent. It is not always diagnosed. Lead is used in some form or other in upward of 110 industries.

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Although lead mining is an old British industry it is not a cause of plumbism owing to the fact that the metal in the ore is in the form of sulphide and therefore extremely insoluble. In this form the ore is known as *galena*. Where the ore occurred as *cerussite*, in other words as an almost pure carbonate, miners have suffered severely in health. This occurred two to three decades ago at the Broken Hill Mines in Australia. Since the cerussite at Broken Hill has all been worked out and nothing remains but galena the occupation of the miner has become healthier.

Smelting of Lead Ore.—The risk of plumbism commences with the smelting of the ore. There are several ways of smelting lead ore, the best known being the open hearth, or Scotch system, and the blast furnace. During smelting, fumes are given off which are rich in lead. In windy weather or when the flues are not drawing well there may be a down-draught so that the men can hardly avoid inhaling some of the fumes. Moreover, in the Scotch hearth system when the molten lead keeps pouring into the receiver or when the slag is being broken up the workman runs the risk of inhaling fumes or dust. The greatest danger the smelter runs is in cleaning out the flues. These are the long passages which carry away from the hearth or the blast furnace the vapor of lead combined with the carbon and ash particles given off by the coal or coke with which the ore is smelted. The fumes may contain as much as 60 per cent. of lead or more in the form of oxide and sulphate. The flues converge toward a high chimney situated frequently a mile away from the smelting works. The object of the length of the flue is to allow the fume as it travels to become deposited, with the view of recovering the lead contained in it. The flues may be underground passages built of brick or wood and be 4 to 6 ft. high so that a man can stand upright in them, or they may be underground galleries built of stone or brick with doors here and there so that men can enter the flues to recover the lead deposited. Formerly, more than in recent years, the fumes which escaped from the chimney stalk being rich in lead not only spoiled vegetation in the immediate neighborhood of lead smelting works but, becoming deposited on pasture land upon which cattle were grazing, caused the death of these animals and led to frequent litigation between farmers and owners of smelting works. It is well known that wild birds are occasionally found dead in the vicinity of such works. The district around Linares in Spain, an important center of the lead industry, has become extremely barren. The goats on the hillsides have suffered considerably; when pregnant they drop their young before term. In consequence of this immaternity there is a difficulty of rearing goats unless they are removed to another locality.

Cleaning out the flues is a dangerous occupation. Men enter them and remove the deposit, but it is a dusty occupation and even though the men when thus employed wear respirators, the fine dust passes through the respirator and makes the men ill. It is dangerous work. The men should not

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be allowed to work in a flue for more than 2 hours at a stretch; they should then be off for 4 hours. The work causes severe headache, creates a disinclination for food, induces retching and sometimes vomiting. If the headache commences when the men are at work it continues all day. It is followed by a sleepless night and frequently also by a rise of temperature. Where the flues are on the slope and water can be added to flush them into large reservoirs wherein the deposit can be silted there is less risk of the men becoming ill. A good illustration of this occurred recently at a large lead works in Newcastle-upon-Tyne. Last year three of the men after cleaning out flues were ill and were obliged to be off work for several days. It was summer weather. After the men had opened the man-holes of the flues and had worked therein for a few hours a violent thunderstorm broke over the city, accompanied by a deluge of rain. For 2 days the rain fell off and on. In cleaning out the flues afterward not one man was ill, owing to the large quantity of water which had entered the open man-holes. It had soaked through the deposit and made the removal of it attended with comparatively little danger to the men. Employees feel that they must recover the dust from the flues for it may contain as much as 30 per cent. of metallic lead. Tons of deposit are removed from flues.

De-silvering of Lead.—The lead removed from the furnace is not always pure metal. It contains silver and occasionally antimony and gold. These, from a white lead manufacturer's point of view, are impurities and have to be removed. Lead crystallizes at a higher temperature than a mixture of lead and silver. When these two metals are present together in the melting pot a crust forms on the surface which contains much silver and little lead. This is skimmed off from time to time and is remelted. On each occasion the crust becomes richer and richer in silver. Sometimes zinc is added to the melting pot as well. The crust which is an alloy of the three metals is skimmed off and the zinc extracted in a de-zincing furnace. Men employed as de-silverers suffer from plumbism. The smelting of zinc is an occasional source of plumbism owing to the lead frequently contained in zinc ore. Spelter, although seldom containing more than 2 per cent. of lead, is a frequent source of plumbism among furnace men. During 5½ years, ending December, 1912, 77 spelter workers in South Wales alone suffered from lead poisoning. Two different types of symptoms are met with in spelter workers: bronchitis and gastrointestinal disorders, which are attributed to the zinc fumes; also nervous affections believed to be due to lead.

Red Lead.—Where red lead is made in the open hearth by raking up the molten metal and allowing air to become intimately mixed with it the resulting product, known as massicot, is a greenish looking substance which becomes yellow or yellowish red on being washed with water. Washing is resorted to in order to remove any unoxidized lead which may be present. After having been thus treated the massicot is replaced in the furnace and kept at a lower tem-

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perature when in consequence of further oxidation its color changes to a bright red. It is then known as minium or red lead. During the raking of the molten metal, fumes escape by the open mouth of the furnace and, although this is always hooded, some of the fumes may be inhaled by the men. In the breaking up and in the packing of red lead, dust rises very freely. There is an opinion that red lead is not dangerous. Such is not my experience. As regards harmfulness there is little to choose between it and white lead. In this opinion I am supported by the experience of Medical Inspector E. R. Stitt[2] of the United States Navy, who treated three seamen who were suffering from saturnine eucephalopathy due to inhaling red lead dust rising from dried surfaces during the act of chipping off of old paint on torpedo boats preparatory to their being repainted. One of the men became insane and had to be taken to an asylum; another after having suffered from colic developed convulsions. All the men recovered. Red lead equally with the carbonate is capable of producing the most severe type of plumbism. In one-fourth of the men who are ill in consequence of having been exposed to red lead the symptoms are usually severe. The making of red lead in closed furnaces and conduits will do much to diminish the number of cases of plumbism due to minium.

White Lead or Carbonate of Lead.—There is still a great demand for white lead despite all that has been said and written against it. The manufacture of white lead in the United States of America was begun in 1777. About 100,000 tons are produced in the States annually. In Great Britain the amount produced is 72,000 tons.

The two principal methods of manufacture are the "Dutch" and "Chamber" processes. In the Dutch process thin perforated plates of metallic lead called "grids or "wickets"" are placed upon the open mouth of earthenware jars containing a quantity of weak acetic acid. These jars are resting on a layer of tan bark and are arranged in rows in a large quadrilateral space one side of which is open. Upon the plates of metallic lead, planks of wood are placed and upon these planks fresh tan is strewn and on the tan another layer of earthenware jars is deposited. By a series of tiers alternating in the manner described the "stack" or "blue bed" is built from the bottom upward. When each tier is completed planks of wood are placed across the open space leading into the stack. The outer doors of the stack are finally closed and kept closed for a little over 3 months. Within a few days of closing the doors the temperature inside the stack rises. This may be partly owing to the activity and reproduction of bacteria within the tan or it may be due to chemical change. The acetic acid volatilizes and, attacking the lead grids, it transforms some of the lead into acetate of lead. While this is taking place, fermentation occurs in the tan and carbon dioxide gas is generated. This reacts upon the lead acetate whereby it is converted into hydroxycarbonate, a basic compound containing two molecules of normal carbonate of lead in combination with one molecule of lead hydrate or lead

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oxide, the formula being $2PbCO_3Pb(OH)_2$. After having been closed for fully 3 months, and when it is believed that conversion of the metallic lead into white lead has become complete, the doors are opened and men enter to remove the "corrosion" as it is called. The process is known as "stripping the white beds," and as there may be considerable quantities of dust raised this particular kind of work is denied to women.

In the Chamber process strips of metallic lead resting on bars are exposed to hot acetic acid vapor, also to carbon dioxide coming from burning coke. The doors of the chamber are hermetically closed and kept shut for from 50 to 60 days. Before the workmen enter a lead chamber to empty it, steam must be injected to render the converted white lead less dusty and therefore less dangerous. The doors ought to be opened also for some hours before the men enter in order to reduce the temperature.

Opinions differ as to the relative values of white lead manufactured by the Dutch and Chamber processes. The latter method of manufacture is more cleanly and the time required for the corrosion of the lead is shorter. House painters who have been in the habit of using white lead made by the Dutch process prefer it. The fact that 66 per cent. of the total quantity of white lead is manufactured by the Dutch process is of itself its strongest recommendation.

Industrial lead poisoning claims fewer victims now than formerly. During 13 years, 1900 to 1912 inclusive, there were reported to the Home Office as having occurred in white lead factories 1393 cases of plumbism with 33 deaths, equal to 2.2 per cent. If the total number of cases of plumbism notified to the Home Office as occurring in all trades be taken, there were during the 13 years already mentioned 8523 cases with 394 deaths, or a percentage of fatal cases of 4.5. During recent years there has been a greater reduction of fatal cases of plumbism among white lead workers than in any other lead process, largely in consequence of the regulations issued and enforced by the Home Office. In 1900 the largest number of cases of plumbism occurred in the white lead trade, viz., 358 with six deaths; in earthenware and pottery manufacture 200 cases were reported with eight deaths. In 1912 the manufacture of white lead no longer occupied the first place on the list. This position was taken by the earthenware and pottery industry. In coach and house painting the number of cases of plumbism has risen largely owing to the prosperity of the motor car industry. House painters suffer from plumbism mostly through inhaling dust given off during the sand-papering of flattened surfaces or from the vapor given off during the burning-off of old paint preparatory to repainting. In the case of persons who have become ill by sleeping in newly painted rooms opinions have somewhat changed as to the cause of their indisposition. Until recently it was thought that the symptoms were those of plumbism, and where a blue line was present in the gums of those who were ill the diagnosis was probably correct. On the other hand the symptoms, severe headache, nausea and retching, developed too soon after

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exposure to paint for lead to be their cause. The symptoms must therefore be due to the inhalation either of the vapor of turpentine alone or combined with emanations from the linseed-oil. Baly of Liverpool attributes the malaise to the inhalation of certain aldehydes given off by lead paints. He found that lead oxide gave off more "unsaturated aldehyde" than lead carbonate. Henry A. Gardner[3] did not find any metal present in the vapors given off by lead-painted surfaces. These vapors contain varying percentages of carbon monoxide and carbon dioxide, to the former of which he thinks the headache, vertigo and retching may be ascribed. Under certain conditions, where traces of sulphuric acid are present, carbon monoxide is given off. In the blood taken from the heart of a guinea-pig, which I had exposed to vapors from a freshly painted surface, the spectroscope showed the presence of carbon monoxide.

The driers which are added to paints are to some extent, therefore, responsible for the symptoms complained of by people who have been living in newly painted houses. Turpentine is a drier, lead is another, so too are benzol, benzine and petroleum spirit. Benzol and benzine vapor causes disintegration of red blood corpuscles, also hemorrhages on mucous membranes, and tender inflamed gums, conditions which recall those observed in scurvy.

The outcry against the use of lead has, particularly in house and ship painting, been followed by the introduction of various substitutes. For leadless paints the demand has increased during recent years. Zinc white is being more widely used and for internal decorative purposes it answers quite as well as white lead, but for external purposes the majority of master painters prefer lead carbonate. They regard its covering properties and enduring powers as superior to those of zinc. Lithopone, which is a mixture of zinc sulphide and barium sulphate, is also coming into repute in house painting. Space will not permit of a comparison being drawn between the various forms of paints. For internal decoration it may be said that zinc paints give all the covering power required and are freer from danger than lead paints, although in one well-known occasion several persons became ill where zinc paint had been used owing to the presence of arsenic, which is frequently present in zinc and which in this particular instance had not been removed after smelting of the ore. The ordinary paints in use have distinctly bactericidal properties. Newly painted rooms are therefore more or less aseptic.

Between 1900 and 1909 there were reported to the Home Office 1973 cases of lead poisoning with 380 deaths of house painters. Paralysis of the hands and fingers is extremely common in house painters. Tancquerel des Planches found paralysis present in 8 per cent. of lead-poisoned French house painters, Teleky in 14.5 per cent. of Austrian painters, and Legge in 22.7 per cent. of British house painters. In the United States it was found that one in every six painters gave a history of having had plumbism in one form or another, in other words 16.6 per cent.

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In 1898 the number of cases of lead poisoning in the China and earthenware trades had been so large as to call for Government intervention. The industry is chiefly located in the district known as the Potteries in New Staffordshire. To the Potteries Sir Edward Thorpe and I were sent by the Home Secretary to enquire into the prevalence of plumbism among the potters and to make such recommendations as seemed desirable. When we visited New Staffordshire in 1898 Sir Edward Thorpe and I found several of the small master potters using glazes for their ware which contained 20 to 30 per cent. of raw lead. Our immediate desire was to reduce the percentage of lead used in the glaze, to have the lead fritted and therefore rendered more insoluble, to keep women away from the dangerous processes and, where possible, to substitute leadless for lead glazes. In a modified form some of these recommendations have been given effect to and will be of distinct benefit to the workers. The percentage of fatal cases of lead poisoning in persons employed in the manufacture of pottery is small; it is only 0.1 per cent. in persons employed in lead processes, in other words 1 in 1000. In the manufacture of earthenware and pottery the work-people most exposed to lead poisoning are the dippers, dippers' assistants, ware-cleansers and majolica paintresses. It is when glaze containing raw carbonate of lead becomes dried, pulverized and raised into the atmosphere that it becomes dangerous.

Space will not permit my mentioning the 111 industries in which Layet, a French physician, tells us that lead is employed. File-cutting need only be mentioned as one of those unhealthy industries, the manufacture of electrical accumulators is another, so too are printing, linotyping, calico dyeing and enamelling of metal plates. In file-cutting and printing other dangers are added. Tuberculosis claims from followers of those occupations a large number of victims. Lead predisposes to tuberculosis by reducing the vital resistance of the individual. Printers and file-cutters run the risk of becoming tuberculous mainly through the presence of tubercular colleagues and the fact that the work is carried on in rooms too highly heated.

Canned food and fruit are sources of plumbism owing to the lead in the solder becoming acted upon by any acid which may be present.

Men employed in a lead factory should neither chew tobacco nor smoke when at work. There is an impression among the men that chewing tobacco is a preventive against plumbism. Considering the opportunities there are of loose tobacco in the waistcoat pocket coming into contact with lead dust, also the fact that it has to be fingered, there is nothing to encourage their impression. Experience points the other way.

CHANNELS OF ENTRANCE OF LEAD INTO THE BODY

Through the skin lead can be, but is infrequently, absorbed. I have seen persons become the subjects of plumbism through using cosmetics and hair dyes which contained lead. The main channels of entrance are the alimen-

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tary canal and the lungs. From an industrial point of view lead in the form of dust is the enemy to be guarded against. When inhaled, lead dust can reach the lungs and be absorbed from the respiratory organs, but most of it is caught in the *nasopharynx* and is swallowed. The experiments of Professor Lehmann of Würzburg and his assistant, Saito, show that when lead dust is inhaled only 12 per cent. reaches the lungs and that 70 per cent. is found in the alimentary canal. When men at work are not wearing respirators much of the dust enters by the mouth and being caught there is dissolved in the saliva and swallowed. On reaching the stomach it is acted upon by the hydrochloric acid of the gastric juice and converted into chloride which is soluble and capable of osmosis. When it has reached the blood it is believed to enter into combination with the proteids and to become lead albuminate, rather an insoluble compound. On the whole, lead carbonate is more soluble in the gastric juice than is lead sulphate. If food is being digested at the same time less lead will be dissolved; hence the desirability of men never commencing work in the morning without first having had food. Lead undissolved in the stomach passes out in the feces in the form of an insoluble sulphide. Some of the lead which has reached the blood stream is eliminated by the kidneys. If not thus removed some of it may pass out into the tissues where, in the form of albuminate, it may remain for months or years doing no harm to the individual, until some change occurs in his metabolism when the lead becomes redissolved and on being reabsorbed causes symptoms of plumbism.

Symptomatology.—One of the earliest signs that a workman is becoming influenced by lead is a gradually increasing pallor. The anæmia, which is fairly quickly progressive at first, reaches a limit and ceases to proceed farther unless it be toward a deepening cachexia. It might therefore be expected that the blood would show on microscopical examination alteration of its structural elements. In a percentage of cases varying from 30 to 70 the red corpuscles or erythrocytes are modified. With any of the Romanowsky's stains several of the erythrocytes show numerous dark dots scattered through their contents. These punctated cells are spoken of as basophiles and when present in the blood in large number the condition is known as *basophilia*. Methylene blue gives satisfactory staining. The best results have been obtained by using a formula recommended to me by Dr. Glibert of Brussels. The fluid is composed of methylene blue, 2 grams; sodium bicarbonate, 12 grams; distilled water 200 grams.

The film of blood when dried is steeped in absolute alcohol or alcohol and ether for half an hour, then dried, afterward steeped for 1 minute in the above solution, removed and washed in distilled water until the color almost fades, when it is dried and is ready for examination.

Ehrlich, Grawitz and others maintain that basophilia is diagnostic of plumbism. Anything over 100 basophiles per 1,000,000 red corpuscles is regarded as absolute proof of lead poisoning. As it is met with in forms of

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anæmia in no way associated with lead poisoning, basophilia is not entitled to this unique claim. When present, basophilia is an important aid in the diagnosis, but it is frequently absent in cases where it ought to be present and it is present where it is least expected. I do not attach to basophilia the importance some physicians ascribe to it nor, on the other hand, do I underrate its value in certain conditions. It has not been found that basophilia bears a direct relation to the length of time workmen have been exposed to lead. In workmen with a well-marked blue line on the gums and with confirmed pallor and who are the subjects of digestive derangements basophilia may be present, but in many instances a most careful examination of the blood fails to reveal more than one or two basophile corpuscles in the whole film. What basophilia is, whether a degeneration or regeneration, and what its meaning, we do not know. In 1031 blood examinations made in the Leipzig Institute of Hygiene, basophilia was present in 187 instances, *i.e.*, in 18.1 per cent. Dr. John Russell has found basophiles in pottery workers varying in number from 100 to 27,000 per million. It is because of other substances, *e.g.*, nitrobenzene and aniline, being able to cause basophilia, also of the presence of basophilia in some forms of anæmia and malaria, that we are disposed to consider the value of basophilia in plumbism as overrated. As opposed to Teleky who reminds us that basophiles are present in the blood of healthy persons, P. Schmidt states that if there are found 100 punctated erythrocytes per million red corpuscles, upon this fact alone a diagnosis of plumbism may be made. At the 1906 Congress of Industrial Diseases in Milan, Biondi declared that he had not found basophile erythrocytes in the blood of persons suffering from severe lead intoxication. On the other hand, Lutoslawsky in 1902 announced that of 107 persons suffering from chronic plumbism only in 17 were there no basophilic corpuscles.

It may be stated that those physicians who believe in basophilia as a sign of lead poisoning maintain that it is in the early stages of the malady that it is most pronounced and that even in this respect there is no uniformity. It is possible that just as there is an individual idiosyncrasy to plumbism, so in those who are becoming lead poisoned the blood of some persons, more than others, may exhibit a greater tendency to basophilia. The change in the red blood corpuscle stained greenish blue by the reagent which has been used reveals itself by numerous dark dots scattered throughout the corpuscles and is not the general bluish coloration of the corpuscle, which is known as polychromatophilia. In the anæmia of lead poisoning there is a distinct reduction in the number of red blood corpuscles. Instead of the normal 5,000,000 erythrocytes per cu. mm. of blood there may be only 3,000,000 to 3,500,000 and with this fall in the number of red corpuscles there is a corresponding diminution in the amount of hemoglobin. Basophilia when present stands in no definite relationship to the amount of hemoglobin. It is detected in the blood of men who are following their occupation in lead works without discomfort and without other signs of plumbism. There is no Certifying

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Factory Surgeon who would on the mere detection of basophilia alone, irrespective of special reference to the extent of it, make it the reason for suspending a workman from the factory. In 70 per cent. of my cases of lead poisoning basophilia was absent. While, therefore, basophilia is an aid to the diagnosis of plumbism its absence is no proof that the malady investigated is not lead poisoning.

In plumbism the white corpuscles of the blood are occasionally increased in the direction of a polymorphonucleosis, but on the whole it may be said that numerical and structural alterations in the leucocytes are not well marked.

The Blue Line on the Gums.—The blue line on the gums is another of those signs which can be of valuable assistance when other signs or symptoms are present, but of itself is no proof of the person being under the influence of lead. The line I refer to is situated at the margin of the gum close to the teeth. In color it varies from a light slaty blue to one of an intenser shade. Although mostly observed on the gums of workmen who are careless in the use of the toothbrush and whose gums are ulcerated, I have found it where the teeth were excellent and where the gums were healthy. It is not necessary, therefore, to have a preceding gingivitis to cause the line, although such a condition of the gums may predispose to it. If the mouths of several workmen in a lead factory are examined, in the majority of them a blue line will be detected on the teeth close to the margin of the gums. This type of blue line is observed in men who are employed in a dusty lead process in the factory. It is not the true Burtonian line but is simply a deposit of sulphide of lead on the teeth, for if the toothbrush is applied or the mouth rinsed with warm water the deposit at once disappears. In the typical blue line described by Burton an actual deposition of lead sulphide has taken place in the cells of the deeper layers of the gum—by cells known as phagocytes. No brushing of the teeth can readily remove this line. It may persist for weeks or months. It is due either to lead which has been dissolved in the saliva or to lead which has been absorbed and comes to the gums by the blood stream, becoming acted upon by the sulphocyanide of potassium present in the mouth, or by sulphuretted hydrogen evolved during the decomposition of particles of food retained between the teeth being taken up by the phagocytic cells of the gums.

A blue line on the gums is of great value in diagnosis when other signs are also present. Even where other signs or symptoms of plumbism are absent it is of importance to note its presence since succeeding events may reveal its origin and meaning. In persons, for example, who are taking large doses of bismuth or who are having fistulous wounds injected with bismuth paste a blue line may appear in the gums practically indistinguishable from that caused by lead. Eliminating such a possibility, persistence of a blue line on the gums after the use of the toothbrush is a sign not so much of lead poisoning as of lead in the system and that at any time, consequent upon altered

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metabolism, other signs or symptoms of plumbism may show themselves. The diagnostic value of the blue line on the gums must therefore neither be overrated nor underrated, since in many cases of undoubted plumbism it is absent altogether. No investigation of a suspicious case of lead poisoning will be complete which did not include an examination of the gums.

Occasionally in addition to a blue line on the gums there may be observed inside the lip or on the inside of the cheek, opposite a decayed tooth or one covered with tartar, a bluish-black patch the size of a three-penny piece or larger. The edges of the patch are irregular. It is due to friction of the mucous membrane against the unhealthy tooth. This patch is confirmatory evidence of plumbism, but only when the administration of bismuth, for example, can be excluded. In copper workers a greenish-blue line is frequently present on the teeth. This line is not, as in plumbism, in the gums. In pyorrhea alveolaris the gums occasionally exhibit a delicate blue coloration, but it is more of a gentle shading than a definite line.

Colic and constipation are early symptoms of lead poisoning. For several days before the onset of abdominal pains a patient may have experienced a disagreeable secretion in the mouth. He may have complained of an unpleasant metallic taste and of an accompanying loss of appetite. Without further warning he is seized with severe pain in the abdomen usually in the neighborhood of the umbilicus. The abdomen is tender to the touch, although in a few instances relief is obtained by pressing the abdomen. The pain is sometimes so severe that the patient is with difficulty restrained. Vomiting may or may not be present. In other instances previous to colic there may be obstinate constipation. The bowels may be closed for a week and opened only with the greatest difficulty by the aid of medicine. Constipation usually attends upon colic, but severe abdominal pain may continue after the bowels have been relieved by medicine, and even where there is diarrhea. During colic Riegel found a high blood pressure which he attributed to constriction of the arteries in the splanchnic area, but in many of my patients during the height of the pain the blood pressure was low, the pulse feeble and slow—only 30 to 40 beats per minute. The secretion of urine falls to 4 and 6 oz. for the 24 hours. The cause of lead colic is obscure. In experimental plumbism I have found the intestinal canal in places so constricted by muscular spasm as to have the caliber completely obliterated. The pain might, therefore, be due to this muscular spasm or it might be the result of an effort on the part of the intestine above the constriction to press onward the contents into the contracted portion below. Colic can hardly be due to constipation and strong peristaltic contraction of the bowel to propel the contents onward, for as already stated the pain persists when there is diarrhea. In the course of 2 or 3 days the severity of the abdominal pain is mitigated but there still remains uneasy painful sensations which become aggravated on pressure, usually worse on one side of the abdomen than on the other. Accompanying this type of pain there is inequality of the radial pulses, inequality of the

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pupils; also pain is complained of when pressure is made along the course of the vagi nerves in the neck. There may be also unilateral sweating of a limb.

Lead colic has been occasionally mistaken for appendicitis and the abdomen opened by a surgeon only to find the diagnosis of appendicitis unconfirmed.

Headache.—When present in plumbism headache is usually severe. The pain is not confined to any particular part of the head. It may or may not be associated with albuminuria. When there is albumen in the urine the headache is probably uræmic, but in a great many persons the urine is quite free from albumen. The headache is toxæmic, none the less, and may be due to lead itself or to poisonous substances formed in the body as the result of the action of lead upon the internal organs. Frontal headache may be neuralgic and associated with arterial spasm. Severe headache is occasionally the forerunner of delirium or convulsions. When convulsions occur in a lead worker they are of grave significance. They may develop without albumen being present in the urine. Convulsive seizures attended by coma constitute what is known as *saturnine encephalopathy*, the most fatal form of plumbism. In females saturnine encephalopathy may be preceded by symptoms suggestive of ordinary hysteria, but the symptoms must not throw the medical attendant off his guard since toxic hysteria frequently gravitates into a more profound affection of the central nervous system.

Albuminuria may be present as an early sign of plumbism during or after an attack of colic and it may disappear, but in large percentage of patients who are the victims of chronic lead poisoning it persists and is accompanied by changes in the blood-vessels and the heart. The arteriosclerosis may be accompanied by an elevated blood pressure. To old lead workers death frequently comes by cerebral hemorrhage and at the autopsy in addition to the cerebral lesions the kidneys are found contracted. Interstitial nephritis is the condition of the kidneys found in the late stages of the disease, but in the early stages and in the more acute forms of the malady the lesion affects more the cells which line the renal tubules. The urine of lead workers frequently contains lead.

In female lead workers menstruation is increased; in those who are pregnant the tendency is for miscarriage to take place. The only possible way for a pregnant female to reach full term is to retire from the work at the earliest possible date. I am disposed to believe that one of the reasons why females are more readily influenced for harm by lead than men is the peculiar action of lead upon the reproductive organs.

Cerebral plumbism has occasionally been diagnosed as tumor of the brain and a portion of the calvarium has been removed to give relief to the headache and pressure. The symptoms are not unlike cerebral tumor: there are severe headache, vomiting and possibly, too, retinal changes. Should the patient who has been the subject of convulsions recover, it may be that the eyesight

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is lost. The loss of vision in lead poisoning is threefold: (1) There are no intraocular changes observed in the retina; (2) the discs are hyperæmic, swollen and mottled and their edges are irregular, the outline of the vessels is obscured and there are retinal hemorrhages; and (3) there are delicate white lines external to the retinal vessels, there are hemorrhages and albumen is present in the urine. In the first form of loss of vision the cause is toxic or there is spasm of the arteries, for vision may be soon regained and no after-effects be left behind. In the second form we have the typical amaurosis of lead poisoning, viz., retinal hemorrhages and swollen discs. Here vision may never be quite regained. When formerly women were allowed to work in the white lead factories saturnine encephalopathy followed by loss of sight was not of infrequent occurrence. Since women have been excluded from the dangerous processes of lead manufacture this severe type of plumbism has almost entirely disappeared. It is not exactly known how the intraocular changes are brought about. Some physicians are of the opinion that lead induces effusion into the subarachnoid space of the brain and thereby causes distention of the sheath of the optic nerve with pressure. In one of my patients who died from saturnine encephalopathy there was found on microscopical examination of the optic nerve round-celled infiltration of the fibrous trabeculæ between the bundles of nerve fibers, the infiltration being most marked at the posterior portion and for some distance behind the lamina cribrosa. In persons dying from acute plumbism with nervous phenomena small hemorrhages were found by Mosny of the Hospital St. Antoine, Paris, in the cortex of the brain. Miliary hemorrhages were found by F. W. Mott in the perivascular sheaths of the small vessels of the cortex and in the substance of the brain in a coach painter who had epileptic seizures. Goadby also found minute hemorrhages at the base of the brain and on the vortex.

Nervous System.—Lead exhibits a predilection for nerve tissue. Not only are the peripheral nerves liable to become affected but the cells of the central nervous system as well. Within recent years, owing to the great improvement in the conditions of labor in lead factories, opportunities for studying the pathology of acute plumbism have become extremely rare. We are familiar with the paralyses of the fingers and hands which takes place in lead workers, but persons thus afflicted seldom die in this stage of the illness. Paralysis of both wrists, the so-called wrist-drop, is characteristic of lead poisoning. Usually both hands are affected, a circumstance which suggests that a general cause such as a toxæmia has been in operation. Occasionally, however, one wrist alone is affected but the other will probably be found to be be paretic. Generally speaking, the paralysis is more marked on the outside of the body the muscles of which are more actively employed. Teleky therefore thinks that fatigue is a contributory cause of the paralysis of plumbism. The influence of previous muscular strain cannot be altogether ignored. It has generally been taught that the loss of power in the muscles in lead paralysis is due to a lesion of the peripheral nerves, an interstitial neu-

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ritis; but Goadby, finding minute hemorrhages in groups of paralyzed muscles of which strain at work may have been a cause, as well as minute hemorrhages in the nerves supplying those muscles, regards the lesion as neuro-muscular.

The common type of paralysis found in lead workers is "wrist-drop." The patient is unable to extend the wrists. The muscles involved are the extensors of the wrists, the interossei extensors of the fingers. The supinators usually escape. This form of paralysis renders the individual extremely helpless. In the brachial type of paralysis the deltoid, triceps, brachialis anticus and supinator longus are affected along with the supra and infra-spinati. In yet another form, the *Aran-Duchenne* type, the muscles affected are those of the outer border of the hand, also those of the thumb including the interossei. Accompanying these there are atrophy and fibrillary tremor of such a degree as to suggest muscular atrophy. Although lead paralysis usually affects the muscle of the upper extremity in correlated groups yet it may be irregular, more on one side than the other; also in children the peroneal muscles and the extensors of the toes may be the only muscles involved. The tibialis anticus as a rule escapes. The peroneal type of lead paralysis may occur alone or it may be associated with wrist-drop. I have seen this form of loss of power in the adult as part of a widely distributed paralysis in which the muscles of the limbs, the back and pelvis were involved. Such widely distributed paralysis is extremely dangerous owing to the possibility of the intercostal muscles becoming affected and of respiration being rendered almost impossible. Fortunately my own patients recovered, but death is known to have come from respiratory paralysis. The muscles of the larynx may become affected, also those of the bladder, but a much more frequent development is paralysis of the muscles of the eyeball causing diplopia.

Persons who are working in lead or who are the victims of plumbism frequently complain of ill-defined pains in the limbs. The pains are spoken of as rheumatic. They may possibly be due to limited patches of neuritis. Occasionally before wrist-drop occurs, pain in the arms is complained of but once paralysis is developed pain is not a common complaint. The reflexes are not uniformily affected. They may be increased; more frequently they are diminished. Tremor of the labio-facial muscles is common and can be usually demonstrated by asking the individual to show his teeth.

In a few male lead workers of 35 to 45 years of age, who have signs of plumbism, there develops an uneven gait, slow and interrupted speech, fibrillary tremor of the tongue and of the nasolabial muscles, defective memory and unequal pupils. The knee-jerks are diminshed or absent. The symptoms suggest general paralysis but there is nothing of the exaltation of ideas nor of the uncertain temper met with in ordinary general paralysis. Saturnine pseudo-general paralysis can be recovered from. This circumstance marks it off from the better known malady. Practically all general paralytics, in consequence of the syphilitic origin of their illness, give a positive Wassermann reaction. Many lead-poisoned patients respond in a similar manner

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but while the Wassermann reaction complicates matters it does not prove that the pseudo-general paralysis of plumbism is of specific origin. Through the kindness of Dr. Irvine, Dr. Shade, Bacteriologist Royal Victoria Infirmary and I have been able to examine several men and women in a lead factory. In our first examination 11 men varying in age from 23 to 58 were taken—4 of them had a well-marked blue line. In 8 of these men the results were positive; in 3 negative. In another series of different workmen, some with blue lines on gums, 4 gave positive results and 7 negative. Of 9 female lead workers 2 gave a positive reaction and 7 negative. In another series of examinations of 8 male workers 3 gave a positive reaction and 5 negative. Of 39 lead workers thus examined in the factory 17 gave a positive Wassermann reaction and 22 a negative, the percentages being 43.6 and 56.4 respectively. It would almost seem as if a slowly developing plumbism and not past or latent syphilis was responsible for the positive reaction in these cases. The absence of a specific history on the part of a patient is of no importance in the diagnosis of syphilis but enquiry into the past with a careful appreciation of the present conditions is a guide. In the largest percentage of the male and female workers who gave a positive reaction syphilis, in my opinion, could be excluded. It is an interesting fact that a lead patient who gives a Wassermann positive reaction may later on return a negative. The precise meaning of a positive Wassermann reaction in a large precentage of lead workers is not yet forthcoming. It must always be borne in mind that lead workers are like other people, not free from syphilis, so that while in some instances a lead-poisoned person is also syphilitic, a circumstance to which the symptoms suggesting general paralysis may justly be attributed, there are others presenting similar symptoms who neither are nor have been the subjects of syphilis.

When and how long after exposure to the metal either in the form of dust or fume, symptoms of lead poisoning will show themselves it is impossible to say. In those patients by whom diachylon has been taken, it is usually 2 or 3 weeks before such a sign as paralysis appears even though the patient may have had colic for several days. Much depends upon the susceptibility of the individual to lead and the amount of dust or fume he is exposed to. Some persons are more readily influenced by lead than others. Females and young persons are more predisposed to plumbism than males and persons of mature years. One of my patients developed signs of lead poisoning 1 week after making electrical accumulators in a small room. Dr. Alice Hamilton found in hospital a white lead worker suffering from colic and neuritis. He had worked in the factory only 3 days. Of 120 patients whom she examined eight had become ill in less than 2 weeks after commencing work, 36 in less than a month and 89 in less than a year.

Colic may occur early in some persons, 1 to 6 weeks after exposure to lead. There is no stated time for signs or symptoms to appear. It is largely a question of individual predisposition. Equally difficult is it to state the

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amount of lead which is capable of creating ill health. All physicians, however, agree that plumbism is much more likely to develop and the symptoms to be more severe when small doses of the poison pass into the body over a lengthened period of time than when only one or two large doses are taken. The system can absorb only minute quantities of lead at a time. When, therefore, one or two large doses are taken, the bulk of the poison is expelled in the feces. As regards drinking water Angus Smith and Professor Rubner tell us that 0.36 mg. of lead per liter of water is the limit of safety. In the famous Orleans case of 38 persons living at Castle Clermont, 13 became ill after having drunk during a period of a few months water which contained 0.2 to 1.5 mg. of lead. Teleky is of the opinion that if 1 mg. or a little more of lead is taken daily for several months, it will cause plumbism. A daily dose of 10 mg. would, in the course of a few weeks, be followed by serious symptoms.

The bulk of industrial lead poisoning is caused by persons breathing fumes or inhaling dust. Kaup found in the air of the drying ovens in a white lead factory, taken at the level of the face of the workmen, 0.134 mg. of lead. Müller found in the air of a lead smelting works 236.8 mg. of lead in 100 liters of atmospheric air. As during a working day of 10 hours 4.5 cubic meters of air could be carried into the lungs there would be inhaled amounts of lead varying from 6 to 25.2 mg. Similarly, as in paint mixing and dry color grinding, there may be 0.178 to 0.25 mg. of lead per 100 liters of air, workmen thus exposed run the risk of becoming lead poisoned. When we add to the above the dust adherent to the fingers and hands, the hair and mustaches, and the clothing and boots of work-people, the opportunities for plumbism are many.

Whether it is absorbed through the lungs or through the stomach and upper part of the small intestines lead enters into close alliance with the proteid of the blood, forming an albuminate, rather an insoluble compound. This remains soluble in a neutral solution but the slightest degree of acidity or of alkalinity as already stated, tends to render it insoluble, a circumstance which explains the inertness of lead albuminate when deposited in the tissues —also the fact that lead which has lain long latent in the system and caused no symptoms suddenly and unexpectedly gives rise to these in consequence of altered metabolism. Since the bulk of lead which enters the body has passed in through the alimentary canal, the liver and spleen after death are the organs which are found to contain it in largest quantities, but lead is also found in the brain, muscles and kidneys.

Diagnosis.—The diagnosis of plumbism may be comparatively simple or, owing to anomalous symptoms, it may be extremely difficult and especially is this the case in industrial lead poisoning where the question of compensation is concerned. Given a history of exposure to the metal, a blue line on the gums and a history of colic with or without wrist-drop there can be little or no doubt about the diagnosis. The cases which are obscure are those

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in which there is no history of exposure to lead and no blue line on the gums but a story of indifferent health of headache and anæmia, with, it may be, paralysis of the muscles of the eyeball. Microscopical examination of the blood may reveal basophilia which, if not due to a recognizable form of anæmia, will be of assistance in coming to a determination. The detection of lead in the urine is of greater importance. It does not follow, however, because lead is found in the urine that therefore the case is one of plumbism for the man or woman may not be ill but it shows that lead is in the system, is being eliminated and that a check to elimination might at any moment be followed by active signs and symptoms of the malady. The harboring of lead within the body or its elimination by the urine and feces in small quantities without the individual being ill places him in the same position as a typhoid carrier. A female lead worker will, for example, bring forth a still-born infant, or a child who dies a few hours after birth, in whose organs lead is found on chemical examination, without herself having shown either signs or symptoms of plumbism. In patients who have become ill through drinking water contaminated by lead there may be none of the ordinary symptoms of plumbism. There may be gradual loss of health, headache, sleeplessness, inability to apply oneself to work, deranged appetite and extreme depression of spirits without any blue line on the gums or history of colic, without basophilia too, and yet traces of lead may be found in the urine. Then, too, it may be that after attacks of recurrent abdominal pain had been dignosed and treated as dyspeptic the individual develops gout and albuminuria which are indirectly the result of the action of lead.

Treatment.—The treatment of lead poisoning is preventive and curative. Drinking water gathered in a peaty soil should have its plumbosolvency tested two or three times a year and dealt with accordingly. Various suggestions have been offered whereby substitutes may be found for lead pipes, but none of them are readily taken up by architects. In lead factories everything possible must be done to prevent the escape of fumes and dust into the atmosphere of the place when work is being carried on. Exhausts should be introduced wherever possible. This recommendation is better than the wearing of respirators. The work-people themselves can be of great assistance by keeping themselves clean, taking warm baths frequently and never touching food without first washing the hands and rinsing the mouth. Females should not be allowed to work in the dangerous processes of any lead industry. Almost the first complaint for which a lead worker seeks advice is colic. Previous to this friends may have observed that such and such a workman was becoming anæmic, but at this stage of the development of plumbism it is rare for medical advice to be sought. Formerly in lead works in the hope of warding off colic employers supplied the work-people with an acid lemonade in which magnesium sulphate was dissolved. It was of doubtful benefit. The good effected was mostly through the beverage acting as an aperient. It gave a false security to the workers. In some lead factories tabloids of calcium

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sulphide, 1 grain, are given almost daily to the men. Whether in consequence of or despite this simple prophylaxis, it must be stated that the men in these factories have kept remarkably well in health.

When colic is severe the patient must be kept warm in bed, if possible, and hot applications applied to the abdomen. As there is usually constipation an enema may be given or a dose of castor oil administered by the mouth. In severe cases of constipation accompanying colic, should the castor oil or any other aperient not have acted, 1 drop of croton oil on a piece of sugar may be given by the mouth. Sometimes the pain is so excruciating that morphine has to be injected. The abdominal pain can be relieved by the administration of sodium mono-sulphate in ½- or 1-grain doses thrice daily. Dr. Stevens of Cardiff has found the internal administration of ¼-grain dose of calcium permanganate helpful.

For colic and constipation a mixture of potassium iodide and sulphate of magnesium with a bitter is much resorted to. Care should be taken in the administration of potassium iodide that the dose to commence with is small, for potassium iodide has the power of dissolving lead lying inert in the tissues, of causing it to be reabsorbed, and as this circulates through the body it may induce a fresh attack of plumbism. To the iodide and magnesium sulphate mixture 5 drops of tinct. nux vomica with a carminative may be added. Massage and electricity should be prescribed in cases of paralysis which are recovering slowly. During epileptiform seizures the bowel should be washed out with mustard and warm water, a tablespoonful of mustard to a pint of water, and afterward an enema containing 30 or 40 grains of bromide should be administered per rectum.

For chronic plumbism and where there are anæmia, albuminuria, headache, loss of vision and œdema of feet the medical treatment resolves itself largely into one of symptoms. It is not absolutely necessary in every instance when albumin has been found in the urine of old lead workers who have no symptoms to suspend these men from employment. Removal from work will not effect a cure. These men live longer and are happier if they are allowed to go to the factory, do light work when there and receive wages for doing so.

In addition to the above another method of treatment can be recommended, especially for lead workers. It is both preventive and curative, known as the Clague-Oliver method—it is an extension of the electrical treatment. The object to be obtained is primarily removal of lead from the body if any be present therein, and secondly the bracing up and stimulating effect of the electricity. The story of the double electrical bath treatment is briefly this: I had produced experimental plumbism in a rabbit. The animal was paralyzed in its limbs. It struck me as the animal had received a considerable quantity of lead by the mouth that if the metal could be removed from the body it might recover. Accordingly I consulted with Mr. T. M. Clague of Newcastle-upon-Tyne, and he suggested that by means of a

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double electrical bath we might succeed in accomplishing this object. Placing the forelimbs of the animal in one bath containing an electrode (positive) and the hind limbs in another containing another electrode (negative), the chest and abdomen of the animal resting on a pad of cotton between the two baths, a current of 15 volts and a milliamperage of 20 was allowed to pass through the animal for a little less than an hour. In the course of a few baths the paralysis had disappeared and the animal was quite well. On chemical examination of the electrodes and the bath-water lead was found. Subsequently lead was administered to the animal and again it became paralyzed. Double electrical treatment soon restored the animal to health again. Several months afterward lead was again given to the rabbit and for a third time it became paralyzed. No bath was given; the animal died. From its spleen and liver lead was extracted. Altogether, in the course of 3 years this animal had received by the mouth 1.096 grains of nitrate of lead, the equivalent of 684 grains of metallic lead. Having succeeded in restoring to health an animal which had been poisoned by lead it seemed that in industrial lead poisoning there was a possible field for the application of the double-bath electrical treatment. I have tried it in many cases with encouraging results. Under its influence the blue line on the gums disappears more quickly than with medicine, paralysis is recovered from more quickly and the duration of colic is shortened. Dr. Patterson of Philadelphia has had unusual success in the treatment of colic by it. As a preventive an electrical bath taken once a week, or at the most twice a week, keeps the work-people in better health than the use of the weekly warm full bath recommended by the Home Office. A lead worker who is absorbing the metal keeps free from symptoms owing to the fact that the daily elimination of lead from his body remains equal or is slightly larger than the intake. The double electrical bath favors elimination. The drawbacks to it are that where the skin is in contact with the salt water there may be slight irritation or itching of the skin; a mild dermatitis may follow. If the bather touches the electrode with his hand or foot there may be painful redness followed by superficial ulceration. It might be thought that if by electrolysis lead can be removed from the body, other metals useful to the economy, *e.g.*, iron, might also be removed. Experiment shows that lead is the more electrolizable of the two metals and that lead is not only the first metal to be removed but that it always comes away in larger quantity than iron. It is desirable to let the work-people have the bath at the close of the day's work since they can go home afterward and take matters quietly for a period. As a preventive the bath should be given for at least 15 to 20 minutes once a week; as a curative agent it should be taken every second or third day. Quite apart from the debatable point of whether lead is or is not removed from the body, the electrical baths have a distinctly bracing up and stimulating influence upon the workers. It is at any rate a line of treatment well worthy of a prolonged trial

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but, like all other forms of treatment, suitable cases, that is, those with not too advanced structural alteration of organs, should be chosen.

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EARLY DIAGNOSIS OF LEAD POISONING

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Early diagnosis, important in every case of ill health, assumes especial importance in those diseases which are preventable, and in which the interest of public health demands that measures be taken to eradicate the conditions which give rise to them. The importance of early diagnosis in communicable diseases has been dwelt upon repeatedly in recent years. In tuberculosis, for instance, we have learned that our only chance of arresting the disease, of restoring the patient to health, and of removing a focus of infection is to recognize the disease in its incipiency. Likewise, the early diagnosis of cases of typhoid fever, scarlet fever, diphtheria and other communicable diseases may call attention promptly to the source of infection and prevent the occurrence of many other cases. With equal force does this apply to occupational diseases. We must learn to diagnose them in their incipiency if we would protect the worker from the more serious manifestations of these diseases, and if we would institute preventive measures against the occurrence of other cases.

Lead intoxication, on account of the great variety of industries in which the metal or its salts are used, is the most prevalent of occupational diseases. While the diagnosis in a well-marked case is usually not difficult, it may not be so clearly defined in the early stages of the intoxication. The tendency among physicians generally is not to make the diagnosis unless the objective signs are found. which are supposed to be pathognomonic of the disease. This tendency to postpone the diagnosis in the absence of certain objective signs is dangerous and often does incalculable harm. The physician who fails to recognize tuberculosis, for instance, without the presence of tubercle bacilli in the sputum very often fails to avail himself of the opportunity of arresting the disease in its incipiency. The same is true of lead poisoning. The waiting for certain signs before establishing the diagnosis may permit the poison to undermine the constitution of the worker beyond any remedial measures, and may delay preventive measures against the source of the intoxication. The objective signs supposed to be pathognomonic of lead poisoning, the presence of one or more of which is still regarded by many physicians as absolutely necessary before a diagnosis can be made, are the lead-line on the gums, basophilic degeneration of the red cells and the presence of lead in the urine and stools.

The technical difficulty of testing for lead in the urine and stools is so great as to preclude such a test from a part of the routine examination in

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general practice. Moreover the presence of lead in the excretions is not constant even in well-marked cases of lead poisoning. On the other hand, lead has often been found in the urine and more frequently in the stools of persons exposed to lead who do not show any evidence of intoxication.

The presence of the lead-line and the basophilic degeneration of the red cells are objective findings which are of practical use in the diagnosis of lead poisoning. As a matter of fact, neither one of these signs is pathognomonic of the disease, and while their presence is a valuable aid in the diagnosis of obscure cases, their absence should not be given undue weight against the diagnosis of lead poisoning when a history of definite exposure to lead can be obtained.

Basophilic granules in the red cells may be found in severe secondary anæmias in which hemolysis has taken place, and are, on the other hand, often absent in cases in which a positive diagnosis of lead poisoning can be made. In looking over a large number of records in which a definite diagnosis of lead poisoning was made in the Out-patient Department of the Massachusetts General Hospital, by the clinical symptoms and the presence of the lead-line, I found that in nearly 50 per cent. basophilic granules in the red cells were absent.

Sir Thomas Oliver states that in only a few instances of the great number of examinations of the blood that he has made, in early as well as in late cases of plumbism, has he found the basophilic granules. Basophilic degeneration of the red cells, in short, does not possess the diagnostic value which some attribute to it, but can at most be regarded as an important confirmatory sign. Still greater importance is attached to the presence of the lead-line.

In my investigation of some of the lead industries of Massachusetts for the State Board of Health, I had occasion to confer with many physicians who, from time to time, treated patients engaged in these industries, and I frequently found that no matter how suggestive the symptoms complained of were of lead poisoning, the diagnosis was not made because of the absence of the lead-line. As a matter of fact, the lead-line presents a great many anomalies, and irreparable harm will often be done when a diagnosis of plumbism is not made because of its absence. In the investigation referred to above, I have frequently seen well-marked lead-lines among workers exposed to lead who did not present, at the time of investigation or at any previous time, any signs of ill health. On the other hand, if one looks into the very extensive literature on the subject, one will find many cases of severe lead poisoning reported in which lead has been definitely demonstrated in the urine and stools, and post-mortem in the tissues, and yet no vestige of a lead-line could be found. Moreover, the lead-line is extremely rare when the gums are in a healthy condition and when the teeth are well cared for. Dr. Oliver fed experimental animals with lead for a long time without observing any lead-line. If, however, an infection of the gums of such animals was produced, the lead-line appeared within a short time.

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The distinction between lead absorption and lead poisoning is not, as a rule, clearly borne in mind. All persons working in lead, unless the most unusual precautions are taken, inhale or swallow some of the poison, but it is excreted through the kidneys and the intestinal tract, or is stored in the body tissues as an insoluble albuminate of lead. If, however, for some reason metabolism is disturbed, poisoning is the result. The presence of the lead-line simply means that the person has been absorbing lead, and is in itself of no greater weight in the diagnosis of lead poisoning than is the fact that the patient's history shows that he is working in a lead process in which we know that he inevitably absorbs lead. A careful inquiry into the nature of the industrial process in which the patient is engaged is therefore of the utmost importance. A history of exposure to lead justifies the diagnosis of lead poisoning in patients presenting obscure symptoms which cannot otherwise be explained. The following cases seen at the clinic of the Massachusetts General Hospital illustrate this point:

Case 1.—A man 45 years old has been a painter for 24 years; 15 years ago he is said to have had an attack of abdominal pain which lasted for 2 weeks. About 5 months ago, after a short period of idleness, he obtained a job painting the inside of refrigerators. He worked for only 2 weeks, and one day while at work he was suddenly attacked with blindness and severe abdominal pain. The pain lasted 2 days; the total blindness persisted for 2 weeks and then he began to improve slowly. At the time of the examination he showed a low-grade optic neuritis and some retinitis of both eyes. The vision of the right eye was one-half and of the left eye two-fifths of the normal. He also had a somewhat contracted visual field for both form and color; examination otherwise was entirely negative. He showed no lead-line, and other than a moderate anæmia his blood was negative. No stippled cells were found. At the time of the onset of his trouble he is said to have had a very marked lead-line.

The history of exposure to lead for 24 years, and the attack of colic 15 years ago which lasted 2 weeks, makes it certain that he had absorbed a considerable amount of lead in his system. The work in refrigerators, without any ventilation, in all probability acted as a predisposing cause, disturbing his metabolism and thereby precipitating the present attack. Even if no lead-line had been observed at the time the blindness occurred, in the absence of lues or any other cause for the optic neuritis, the diagnosis of lead poisoning is perfectly justifiable.

Case 2.—A man of 50, a painter for 30 years, is said to have had "painter's colic" 12 years ago which lasted for 4 or 5 weeks. Two months previous to his present illness he had to sand-paper old paint a great deal, and was thus exposed more than usually to great quantities of dust. About 3 weeks previous to his coming to the clinic he felt a sudden stabbing pain in the left elbow which lasted only for a few minutes. His left arm then began to feel stiff and weak, and he had a continuous dull pain in the elbow-joint. Physical examination was entirely negative except for slight weakness of the flexor muscles of the forearm. There was no lead-line and no stippled cells.

In this case, likewise, the history of long exposure and the absence of any other condition to explain the symptoms makes a diagnosis of lead

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poisoning justifiable; a subsequent examination revealed the presence of some stippled cells.

The history of exposure is the all-important factor in the diagnosis of plumbism.

In cases of non-industrial plumbism, however, in which no history of exposure can be obtained, the presence of the lead-line or of the basophilic granules is of inestimable value in the diagnosis, since it offers a clue for the explanation of certain symptoms which would otherwise be obscure.

The following case reports taken from the records of the Massachusetts General Hospital will illustrate the reluctance, even among good clinicians, to make a diagnosis of plumbism in the absence of the lead-line or stippling:

Case 3.—A man of 36, a sheet-metal worker for 11 years, handles a great deal of lead in his work, complains of pains in the abdomen, loss of weight, weakness of extremities and attacks of diarrhea followed by constipation. No lead-line.

Case 4.—A man of 34, a painter for 10 years, complains of attacks of colic, headaches, obstinate constipation and general nervousness. No lead-line, no stippling.

Case 5.—A man of 22, a painter for 4 years, complains of constipation, attacks of severe abdominal pains and headaches. He looks pale; no lead-line, no stippling.

Case 6.—A painter of 22, who has worked at the trade for 10 years, complains of obstinate constipation, pain in his joints, headaches, numbness and weakness of extremities; he is easily fatigued. No lead-line or stippling found.

Case 7.—A worker in a rubber factory for 6 years complains of abdominal pains, obstinate constipation, severe pains in lumbar region, loss of appetite, and general nervousness. No lead-line found.

Case 8.—A man of 49, a painter for 25 years, gives a history of lead poisoning when he first started his work. He now complains of tremor and weakness of hands so that he frequently drops his brush while at work. He has attacks of abdominal pains, pains in his joints, and is pale. No lead-line or stippling found.

Case 9.—A man of 38, who worked in a car factory for 14 years, and part of whose work consisted of mixing lead, complains of sharp pains in the abdomen, attacks of dizziness, fine tremor of hands, metallic taste in the mouth, and pains in the extremities. He lost considerable weight. No lead-line or stippling found.

In none of the foregoing cases was a definite diagnosis of lead poisoning made. There is no doubt, however, that if a lead-line was present in any of these cases or if stippled red cells were found, even if the symptoms had been less characteristic, a positive diagnosis of plumbism would have been made. Yet the occupation of these patients was sufficient evidence that lead absorption was taking place, and the symptoms were sufficiently characteristic to warrant a definite diagnosis without the corroborative evidence of the lead-line or stippling.

In consequence of the undue weight which is given to the objective signs, persons who show themselves, by the symptoms of which they complain, susceptible to the influence of lead are permitted to continue at their work without any additional precautions and without any warning as to the danger to which they are exposed. This is shown by the following illustrative case reports, taken from the records of the out-patient department of the Massa-

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chusetts General Hospital. These patients attended the clinic for a long time, and no diagnosis of plumbism was made because of the absence of the lead-line or stippling. At some later date, however, although the symptoms were the same, the diagnosis of plumbism was made because of the appearance of one or the other of these objective signs.

Case 10.—A worker in a rubber factory came to the clinic complaining of precordial pains, headaches, pains in the arms and attacks of abdominal pains. He had evidence of mitral regurgitation. There was no lead-line, and no diagnosis of lead poisoning was made. He came again 8 months later with the same symptoms but of increased severity. A lead-line was found present at this time, and a diagnosis of plumbism was made.

Case 11.—A painter of 23 came to the clinic, November, 1907, complaining of pain in the back, numbness and weakness of extremities, abdominal pains, headaches and nervousness. No lead-line was present, and no diagnosis was made. In 1912 he presented himself with the same symptoms. Lead-line and stippling were found, and a diagnosis of lead poisoning was made.

Case 12.—A painter presented himself at the clinic in July, 1907, complaining of constant severe pain in the upper abdomen, attacks of vomiting, pains in the knees and dizziness. A diagnosis of arteriosclerosis was made. In July, 1913, he presented himself again with the same symptoms. A lead-line and stippling were found. Diagnosis, lead poisoning.

Case 13.—A machinist of 28 came to the clinic in 1906 complaining of attacks of severe abdominal pains, bad taste in the mouth, obstinate constipation, attacks of nausea and vomiting. He came many times during 1910 and again in 1911, complaining of the same symptom-complex. In December, 1911, a lead-line was found and many stippled cells. Diagnosis of lead-poisoning was made.

Case 14.—A worker in a rubber factory, aged 57, came in 1904 complaining of pain in the cardiac area and in the right shoulder, pains in hips and legs, weakness of extremities, dizzy spells, easily tired and constipated, also wasting of extensor muscles. No lead-line was found (patient had false teeth). No diagnosis of lead poisoning was made until 1905, when lead was found in the urine.

Case 15.—A carriage painter of 38 came in 1903 complaining of pains in the back between the shoulder blades. There was no record of lead-line or stippling. No diagnosis was made. Later, in the same year, he came complaining of attacks of dull pain in the region of the umbilicus, and sharp pains in the lumbar region which kept him awake for 2 nights. A history of alcoholic excess was obtained for the last 2 weeks. No lead-line was found. Diagnosis, alcoholism. In 1908 he came again, complaining of pains in his back and in his feet, general weakness and nervousness; no diagnosis was made. Several weeks later he came again complaining of the same symptoms. A lead-line was present at this time. Diagnosis of lead poisoning was made.

The unwarranted delay so frequent in diagnosing plumbism even in the presence of a definite history of exposure to lead is well illustrated by the last case. For nearly 5 years this man has been presenting himself at various times at the hospital, complaining of a train of symptoms which, taken together with his work, should have made a diagnosis possible, and yet he was permitted, during all that time, to continue his work without any special precautions until the lead-line appeared, when the diagnosis of lead poisoning was made.

It would, of course, be folly to attribute to lead all the ill health of lead workers. The symptoms, therefore, have to be considered carefully before

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the responsibility of the diseased condition is laid to the industry. The symptoms of early plumbism are not well defined, yet there is a group of symptoms which, when not open to explanation on any other basis, must, when a history of exposure to contact with lead has been obtained, be taken as evidence of early lead poisoning. Indeed, it is important to inquire carefully as to any possible industrial exposure to lead whenever certain ill-defined states of ill health present themselves among workers, when such diagnosis, for instance, as anæmia, debility, constipation, lumbago or chronic arthritis, is made. Inquiries as to the details of the patient's work should be made, for unless the physician has a familiarity with the various industrial processes, the general designation of the patient's work may not indicate any exposure to lead.

Pallor of the skin, muscular weakness, rheumatic pains, loss of appetite, constipation, or constipation alternating with diarrhea, abdominal pains, general nervousness, and persistent headaches should always be regarded seriously when occurring in persons exposed to lead, for they may be the precursors to the more serious nerve lesions, such as wrist-drop, encephalopathy, and to the more insidious and less dramatic changes in the vascular and excretory systems, that of arteriosclerosis and chronic nephritis.

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LEAD POISONING IN THE UNITED STATES

BY ALICE HAMILTON, M. D., Chicago, Ill.

During the last five years several State and Federal investigations have been made to determine the prevalence of industrial lead poisoning in the United States. These investigations do not lead us to believe that lead poisoning is a slight evil in our country; on the contrary we are forced to admit that in most industries our rate is higher than the German or British in the same trades. Our methods are often dangerous and our factory sanitation, though in some places excellent, is in other places distinctly bad. Nor are the living conditions of American workmen better, their food more abundant, their housing superior to those of British or German workmen in certain lead trades. In skilled trades such as painting, plumbing, printing, white ware glazing, wages are high and the men live well, but in some of the most dangerous lead trades, the making of white lead or the oxides, the smelting and refining of lead, the making of storage batteries, the painting of manufactured goods, the employees work for wages which are below the sum required for a family to maintain what is called an American standard of living.

Lead Poisoning in Illinois.[1]—This is a fairly typical state with such lead industries as smelting and refining, the manufacture of white lead and the oxides, paint grinding, sanitary ware enamelling, tile glazing, litho-transfer works, as well as all the ordinary industries in which metallic lead is used. The inquiry made by the Commission on Occupational Diseases covered 3 years, 1908 to 1910, and 578 cases of lead poisoning were found to have occurred during that time, 308 of them in the last year and chiefly in the city of Chicago. They were scattered through about 70 occupations, some of which had never been supposed by Chicago physicians to involve risk of lead poisoning. Among the less familiar ones were: the making of car seals, of tin foil, the wrapping of cigars in tin-foil, laying lead coated electric cables, making artificial flowers, handling wall paper colored with lead pigments.

Inquiry showed that several cases which were reported as brass poisoning were really lead poisoning and that the brass industry in Chicago was responsible for many cases of plumbism. Litho-transfer works sent several serious cases to the Cook County Hospital and owing to the general ignorance concerning industrial processes some of these were not recognized at first but were treated as appendicitis. The same thing was true of another industry which is very rarely mentioned in the literature of lead posioning, the work of the commercial artist or retoucher. Several cases of supposed zinc poisoning were reported to the Illinois Commission, the victims being

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commercial artists who used what they thought was a zinc paint in making sketches for catalogues and advertisements, and in retouching the high light in photographs for the same purpose. Evidently several of them had been treated by physicians who did not suspect lead poisoning, but the supposedly zinc white paint proved to be white lead and the men's habit of bringing their brush to a point by sucking it explains the poisoning, as does also the use of the paint in the form of a very fine spray. Fifteen such cases were found among the members of the profession in Chicago.

Another unexpected source of lead poisoning proved to be the zinc smelting industry in La Salle County. The ore contains less than 1 per cent. of lead, it is said, but enough is volatilized to give rise to plumbism among the smelters. Tinsmiths, makers of coffin handles, copper and nickel buffers, sorters in junk shops, and men who finish cut glass by rubbing it with a lead putty powder, were others of the miscellaneous workers who made up the Illinois lists.

Sixty Fatal Cases of Industrial Plumbism in New York State in 1909 and 1910.[2]—Histories of all the reported deaths from industrial plumbism in New York State during the years 1909 and 1910 were collected and published by John B. Andrews. Forty-five of the 60 cases recorded, or 75 per cent., had worked with wet paint, three had worked at lead smelting, four in the printing trades, three in white lead works, two in storage battery plants, and one each in tinning, in making rubber balls and in making lead pipe. As would be expected, nearly all of them were chronic cases, of several years' duration. Two cases, on the other hand, developed fairly rapidly. One of them, a painter, had worked at inside painting only 11 months during which time he had had cramps in his stomach during several months, but the final attack of "gastro-enteritis and chronic lead poisoning" lasted only 2 days. The other was a boy of 18 years who, some 17 months before, had gone to work in a comparatively safe lead trade, the making of tin pails, but it was carried on in a poorly ventilated shop. At the end of 6 months he felt ill and began to lose weight, at the end of the year he was forced to leave work and after an illness of 10 weeks died of "chronic lead poisoning."

Among the more unfamiliar occupations in this series of 60 fatal cases were: The making of rubber toys and balls, the packing of machinery covered with wet paint, portrait painting, and drawing bristles through the pads of brushes which had been painted with white lead paint.

Lead Poisoning in the City of New York.[3]—A survey of the lead trades in the city of New York and a report on cases of lead poisoning discovered by the investigators and occurring during 1909, 1910, and 1911 was made by Edward Ewing Pratt for the N. Y. State Factory Investigating Committee. The report includes a detailed description of some of the more important industries: The manufacture of white lead and red lead, the making of paints, the use of lead as a hardening and tempering agent. This last, an occupation hardly mentioned in most of the works on lead poisoning, Dr. Pratt

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found to be very dangerous. He traced no less than nine cases of lead poisoning to one factory which had been in operation only 1 year and in which the regular force employed was only nine men. The work involves hardening steel magnets in a bath of molten lead the temperature of which in this case was from 1400° to 1800°F. Here they remain till red hot, when a workman takes them out and plunges them into a barrel of water to cool. Then they are stacked about an upright bar and rubbed with sandpaper to remove adhering bits of lead. In addition to the risk from fumes and from the lead dust caused by the rubbing there is dust from the oxide which forms on the surface of the molten lead and is skimmed off and thrown on the floor. A similar process was seen in a factory where piano wires and springs are made.

Lead was found to be used in the making of linoleum and oilcloth, first as a drier added, in the form of litharge, to boiling oil, second in the paints which consist largely of lead colors. Six cases of lead poisoning were on record in the hospital nearest to the village in which the factory is situated.

Dr. Pratt studied 109 of the 376 cases discovered by his investigators. Of these, six were fatal, 20 had wrist-drop, 18 paralysis of other limbs, 34 incipient paralysis, and two encephalopathy. The painting trade was most largely represented, with 42 cases, then came white lead with 23, and then work with solder and other metallic lead. Among the less usual sources of lead poisoning in this list are such industries as stamping designs on embroidery with a mixture of rosin and white lead; chipping old red lead paint from the hold in a battleship; polishing diamonds, the diamond being embedded in a pear-shaped lump of lead and held against a revolving disc, which at the same time grinds off part of the lead.

Very interesting and valuable tests to determine the presence of lead in the air surrounding lead pots, casting machines, soldering benches, etc., are described by C. T. Graham Rogers and John H. Vogt.[4] The results are of great value in settling the question, which is still in dispute as to whether or not lead fumes are given off under certain circumstances. For instance, when I was investigating storage battery plants, I was always assured that there could be no danger from the casting, because the lead in the kettles was below the fuming point. I found cases of lead poisoning nevertheless among moulders and the explanation is furnished by the tests of air in two casting rooms made by Rogers and Vogt. They found in 1 cubic meter of the air of a casting room 3.4 mg. of lead, and in another factory where the kettles are covered with hoods 1.0 mg. As an adult breathes about 4.5 cubic meters in the course of 10 hours, this means that it would be possible for a man in the first factory to breathe 15.3 mg. during one working day and for a man in the second, 4.5 mg. provided this contamination of the air were constant. Another striking report on the amount of lead in the air is found in the description of a factory making dairy implements. A number of cases of

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lead poisoning had been reported among the men engaged in making cream separators, and when a visit was made to the plant it was discovered that during the finishing process a putty consisting of 65 per cent. lead was put on the metal part, baked in an oven and when hard, sandpapered. An analysis of the air in the room, which was large and well ventilated, showed no lead but a sample taken at the breathing level of a worker while sandpapering showed 68.8 mg. of lead per cubic liter of air, clearly proving that the danger came from inhaling the dust laden air during the process of sandpapering.

A man in a factory where brass nozzles are caulked into hydrants by means of molten lead was heating his lunch at the lead pot. The air near this pot at the breathing level had 5 mg. of lead in 1 cubic meter.

Some negative results also in this inquiry are interesting. We read in foreign works about lead poisoning in the making of glass, in dyeing, in the manufacture of shoes, in harness making and in polishing precious stones. Rogers and Vogt found only two out of six glass factories using lead, but in those two, dust is allowed to escape and the air of the mixing rooms is badly contaminated with lead. In other industries, dyeing, shoemaking, etc., there was found to be no danger from this source. On the other hand, the mixing of ingredients for rubber (lead oxide and lead chromate are used) was found to be attended with danger, as no precautions are taken to protect the weighers and mixers from dust. A sample of air taken in a mixing room during the process of filling the mixer yielded 8 mg. of lead per cubic meter.

These writers advise that special rules be passed by the State for the following industries: Manufacture of white lead; of dry colors; of lead oxides; of paint and varnish; of storage batteries; potteries; smelting, making of alloys and plumbers' supplies; manufacture of articles from metallic lead; manufacture of rubber goods where lead is used; painting of manufactured articles.

The inquiries carried on by the Bureau of Labor of the Federal Government and more recently by the Bureau of Labor Statistics of the Department of Labor,[5] have concerned individual industries and the method has been to make an intensive study of the establishments in which the work is done and to collect all the available information about lead poisoning in each one. This information is always far from complete. There are now many states which require that all cases of industrial lead poisoning be reported to some central office, but most of them have passed such a law only recently and have as yet no really trustworthy statistics. Moreover American physicians are not yet convinced of the necessity of this legislation and the majority do not take the trouble to send in reports of any but the severer cases, while some who are employed as company physicians feel that they are bound in loyalty to their employers to keep secret the number of cases of plumbism which occur in the plant. I have myself seen a bill presented by a doctor to the company

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asking pay for the treatment of 57 cases of acute lead poisoning, while his report to the State put the number at 27 only.

This makes it impossible in all but a few instances to know exactly how much lead poisoning there is in any of the lead trades in the United States and the cases discovered in the course of these inquiries always fall below the truth. This was especially true of the first investigation, that of the white lead and lead oxide industries, for at that time the subject of lead poisoning among working people had attracted little attention and there were many employers who were genuinely ignorant of the extent of the evil in their own establishments. The only study I have been able to find in American medical literature of industrial lead poisoning among white lead workers is an article by Wm. R. Hobbs,[6] written in 1898. He reports 42 cases from one white lead works, all but eight of them severe in type. Two had epileptiform attacks, one was a case of acute meningitis, lasting 2 weeks and ending in recovery, a fourth had complete paralysis of biceps and deltoids, dilated heart with mitral regurgitation, parenchymatous nephritis, later on, insomnia, loss of memory, listlessness, œdema, anasarca and death. Some of Hobbs' cases developed after only a few weeks' exposure, and no less than 35 of the 42 after less than a year's exposure.

Manufacture of White Lead, Litharge, Orange Mineral, and Red Lead.[7]— The white and red lead industry is carried on in Brooklyn and Staten Island, N. Y.; Camden and Perth Amboy, N. J.; Philadelphia, Pittsburgh and Kensington, Pa.; Cincinnati, O.; Chicago, Ill.; St. Louis, Mo. and Joplin, Mo.; Omaha, Neb.

There are also two establishments on the Pacific coast but these were not included in this investigation. The regular pay roll of the 23 factories east of California was in 1911 about 1600 men, but a much larger number than that was employed during a year's time, for the superintendents all admitted that the force was very shifting. In one establishment 250 men had been hired during 6 months in order to keep up a force of only 58 men.

The processes used in making white lead in the United States are four, the Old Dutch, the Carter, the Matheson and the Mild, but the first is the most usual and the last two are used in only one factory each. For the Old Dutch process, the lead is cast in thin discs and these are packed in pots with acetic acid and buried in old tan bark for about 100 days, when the metallic or "blue" lead is found to have "corroded," that is, changed to white lead, the basic carbonate. In removing this white lead ("stripping the white beds") there is a great deal of dust and the men cannot sprinkle it with water as is done in England and Germany, because in our factories the products of the corrosion must go through a series of grinders and screens to separate the white lead from the unchanged metal in the center of the disc, and dampness would result in clogging the screens. In the factories I have visited in England and Germany this separation is done by washing the corroded lead off in great troughs of running water. A Belgian factory, near Brussels, had

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a process similar to the American. This use of dry instead of wet separation increases very much the difficulty of making American factories dust-free, and at the time this investigation was made the work of "stripping" was productive of a great deal of poisoning. Since then, however, I have seen in three plants an excellent protective device, a truck with a cover which has an opening just large enough to allow the pot of white lead to be emptied in. Another opening is for a large exhaust pipe which carries off the dust formed by the falling of the contents of the pot. This exhaust pipe is flexible and long so that the truck can be moved about without disturbing the mechanism.

The second danger spot in a white lead establishment is the dry pan room, where the white lead after repeated washings is pumped into great hot pans and left to dry, when it is emptied into trucks and taken to the packers or to be ground in oil. In most establishments visited, a certain amount of white lead was ground as "pulp lead," the oil displacing the water without any drying process. Work as conducted in dry pan rooms was dangerous work when first investigated. I remember seeing two men in a low, hot pan room, standing on the dry white lead in the pan and shovelling it to the edge, then throwing it into a truck below as carelessly as if it were so much sand. Since then, however, this work has been much improved in the better factories, the pans entirely enclosed, connected with the exhaust system, and the dry lead emptied into a screw conveyor which is placed inside the enclosing jacket, so that the dust raised by the shovelling does not escape but is drawn in by the suction of the exhaust.

In the Carter process, the dust problem is a serious one, for this is a quick corrosion of lead which has been reduced to the finest powder, so that from start to finish the men are handling powdered material. The advantage of the method is that it requires a small force of workmen and the modifications made from year to year tend to lessen still further the amount of hand-work.

The Matheson process is a precipitation process and safer than the Old Dutch, because work in the corroding stacks is replaced by precipitation of white lead in water. The Mild is also a wet process, atomized lead being exposed to air and carbon dioxide and water in large cylinders. However in both these factories the drying and grinding and packing of the finished product is as dangerous as in an Old Dutch establishment.

The roasting of lead oxides is not carried on in connection with lead smelting in the United States as it is in most other countries, with the exception of one smelting plant. It is done either separately or in connection with the making of white lead. The dangers in this work consist in the fumes from the furnaces, and in the dust from dumping, grinding, screening, and packing the oxides. I have seen some red lead factories in this country which were so free from dust that one would not guess from their appearance that colored materials were made there. Mechanical furnaces, well hooded, have done away with fumes, exhausts over the dumps and closely covered grinders and screens have prevented the escape of dust. In such plants the danger of

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lead poisoning is slight, but in a poorly managed oxide works, with much hand-work, the danger may be even greater than in the white lead department. In one factory I found that during the preceding 16 months there had been 14 cases of lead poisoning among 65 white lead men making a rate of 22.5 per hundred, and 7 among 12 red lead men, or 58.3 per hundred. The average period of employment was 4 years for the white lead men and only 1 year for the red lead men. It is partly the great fluffiness and lightness of the oxides, especially litharge, which makes them so dangerous, for their solubility in human gastric juice is less than that of the basic carbonate.

The number of cases of lead poisoning found in these two industries in 1911 was 388, occurring during 16 months' time in factories with a pay roll of 1600 men. Neither of these figures is accurate. More men than 1600 were employed in these plants during that time, and more than 388 cases of lead poisoning undoubtedly occurred, for this number represents only those who could be traced by investigators working against great difficulties. The fatal cases numbered 16. The period of exposure before poisoning occurred was usually short, 89 out of 120 falling ill after less than a year's work. Undoubtedly there is less lead poisoning now in this industry than at that time, for, in addition to the many improvements which have been made in the factories, physicians have been employed to examine and treat the men, but the falling off in the sickness rate cannot be statistically proven, since these physicians, if they report cases of lead poisoning at all, report only the severer ones.

Glazing and Decorating White Ware and Sanitary Earthenware and Tiles.[8]—The pottery industry in the United States is carried on chiefly in two districts, the region around East Liverpool, Ohio, which includes towns in West Virginia, and in and about Trenton, N. J. White ware includes table and toilet ware; sanitary earthenware includes tubs, basins, closets, sinks, etc., which are made of glazed clay, in distinction from porcelain enamelled iron ware.

In these potteries there are about a thousand persons employed in work which involves exposure to lead in some form, only 150 of them being women. No leadless glaze is used, except on the very large ware, the bath tubs, laundry tubs and sitz baths, which are covered with a leadless glaze and subjected to one prolonged firing that at once biscuits the clay and fuses the glaze. Smaller pieces, closets, wash-basins, and all table and toilet ware, are first fired to biscuit the clay and then covered with a lead glaze and fired again. The glazes used in the 30 table and toilet ware potteries investigated contained lead in proportions ranging from 5 per cent. to 20 per cent., but in only seven was it as low as 5 per cent.; in the other 23, the lead was from 15 per cent. to 20 per cent. For sanitary ware less lead is used, seven out of nine potteries using a glaze with less than 15 per cent. lead. White lead is the compound almost always used.

In England and on the Continent there has been for many years a strong

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movement in favor of insisting on the use of "fritted" glazes. In fritting, the lead is added at the beginning to the other glaze-forming constituents and is fused with them, in the course of which a large part of it is changed from a soluble to an insoluble form, the disilicate. The statement has frequently been made to me in American potteries that the glaze in use has all been fritted, but unfortunately it is the American custom to add part, if not all, of the lead after fritting.

The occupations which expose pottery workers to the danger of lead poisoning have to do with the glaze and with the lead colors used in decorating. Mixing the glaze is done by laborers under the direction of a skilled foreman. Dipping the ware in liquid glaze is skilled work and is always done by men, as is also the stacking of the dipped ware in the kilns for firing. The dippers' helpers, who clean the excess of glaze from the ware, are unskilled women or boys, or in sanitary ware potteries where the ware is very heavy, young men. Decorating is skilled work and employs both men and women. Most ware in this country is now decorated by means of litho-transfer paper, which is entirely devoid of danger, but there is still some color blowing and hand decoration, though chiefly in art pottery, not white ware.

In none of the potteries visited were proper washing facilities provided for the people exposed to lead, nor lunch rooms, nor working clothes, nor was there any evidence of an effort on the part of the management to protect the work-people from lead poisoning. Even in newly erected potteries the floor of the dipping room was always of wood and dry sweeping was very common. I saw the dippers' helpers in several places prepare for lunch by rinsing their hands in a pail of water which the dippers had already used, and then sit down and open their dinner pails in that very room. They say that in the 2 years that have elasped since this investigation was made much has been done to improve conditions in the industry, but very radical changes in construction and management would be needed to bring our potteries up to the British standard.

White ware potters are an intelligent class of workmen with a strong trade union, good wages, and short hours. There are, however, in these same potteries laborers and boys and women and girls who earn low wages and do not have any of the advantages of organization. It was among these last that the highest rate of lead poisoning was found. Even among the dippers and kilnmen the rate was high. One dipper's local of 85 men had had in 1 year 13 cases of lead poisoning, as many as there were among 786 English male dippers, making a rate for our dippers of one in six or seven and for the Staffordshire dippers of one in 60 or 61. Though it is a skilled and well-paid trade 119 dippers averaged only from 17 to 18 years employment and 200 glost-kilnmen only 14½ years. As for the dippers' helpers, the rate of lead poisoning among them was found to be 13 in 180 in Trenton, where men and boys are employed, and 29 among 150 in Ohio, where women are employed. This greater incidence among women is always noted by

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English authorities, especially by Oliver. Comparing the dippers in East Liverpool with their women helpers we find the men's rate to be 17 per 100 employed, the women's 33 per 100, almost double.

Art and Utility Ware.—This term covers the yellow earthenware used in kitchens, the dark brown teapots known as Rockingham ware, and majolica or decorated ware of all kinds, vases, pedestals, umbrella stands, spittoons, etc. All these are made with a glaze rich in lead, containing from 20 per cent. up to 50 per cent. In these potteries there is much more mixing of glaze and much more decorating than in the white ware. Decorating is done partly by hand painting partly by spraying on the colors by means of an atomizer and a stream of compressed air. This is the process which in British reports is spoken of as "color blowing," and regarded as requiring special safeguards. The ware is held under a hood furnished with an air exhaust and if the draft is strong enough, the spray not driven with too much force, and the hood deep enough, and the decorator's sight good enough so that he can hold the ware at a distance from his face, the work is not dangerous, but in many American potteries these conditions are not found. Poor hoods and feeble exhausts were found to be quite common.

The workers in this class of potteries are exposed to greater dangers than those in white ware, because the glaze is richer in lead and because more color blowing is done, and in addition it was found that the standard of cleanliness is even lower in these potteries than in the white ware branch, so that the exposure to lead dust is greater. Another great disadvantage in this branch of pottery work is the fact that it is entirely unorganized and consequently wages are low and the proportion of women employees is much higher. The influence of all these factors can be seen in the following table which gives the rate of lead poisoning among the white ware potters and that among the art and utility ware potters. The men only are included.

White ware:
No. employed, 796. Cases in 2 years, 60. Rate per 100, 7.5.

Art and Utility Ware and Tiles:
No. employed, 304. Cases in 2 years, 63. Rate per 100, 27.

Tiles.—The glazing and decorating of tiles is carried on in the same district as the making of art pottery, the region around Zanesville, O., and as it is also a dangerous and insanitary and underpaid industry it was found impossible to separate the cases contracted in the potteries in this district from those contracted in tile works and the two industries had to be studied together, though in many ways the work in a tile factory is different from that in a pottery. Aside from the Zanesville region, tile factories were found in Trenton, N. J.; Newell, W. Va.; Covington and Newport, Ky.; Indianapolis, Ind.; Chicago Heights, Ill.; and Morristown, Pa. The glazes in these plants contain as little lead as 5 per cent., in the case of white glaze, and as much as 50 per cent. or even 60 per cent., in colored glazes. The lowest quantity,

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5 per cent. was found in only two factories; in all the others the lead content ran from 20 per cent. up. White glaze is applied by machinery in some factories, but colored tiles are glazed by dipping or by pouring on the glaze, or in the case of mottled "onyx" tiles, by scattering it from a pail by hand.

Scraping off the excess of glaze, or "fettling" as it is called, is more dangerous than actual glazing because it is dustier. In all English tile works, and in many German, it is the rule to scrape the excess glaze while it is damp and let it fall into a pan of water. In all the tile works I have visited in this country, much of the fettling, if not all, is done dry and the glaze dust allowed to fall anywhere. I have even seen girls brush off glaze with a stiff brush, and then blow the tile clean. Nowhere have I seen any apparent recognition of the danger of this work.

The rate of lead poisoning among men and women in these two branches is given in the following table. It will be noticed that here where there is no advantage on the part of the men, where all earn low wages, the men have a somewhat higher rate than the women, but then they remain longer in the industry than most of the women do.

Males,	No. employed, 304.	Cases of plumbism in 1 year, 48.	Rate per 100, 15.1
Females.	No. employed, 243.	Cases of plumbism in 1 year, 28.	Rate per 100, 11.5

The full effect of the neglect of all hygienic precautions in this industry can be seen if we compare our record for 1 year with a year's record in British potteries, which are better managed than any I have visited in other countries. This table shows the contrast.

FREQUENCY OF LEAD POISONING IN EACH SEX, IN ALL POTTERIES, GREAT BRITAIN, 1910, AND IN POTTERIES VISITED, UNITED STATES, 1911

Sex	All potteries, Great Britain			Potteries visited, United States, 1911		
	Employees in 1907	Cases of lead poisoning, 1910	Ratio of cases of lead poisoning to number of employees	Employees	Cases of lead poisoning	Ratio of cases of lead poisoning to number of employees
Males	4504	40	1 to 113	1100	87	1 to 12 or 13
Females	2361	37	1 to 64	393	57	1 to 7
Total	6865	77	1 to 89	1493	144	1 to 10 or 11

Porcelain Enamelled Sanitary Ware.[9]—The very heavy, solid bath tubs which are found in large houses and in hotels are made of earthenware and covered with a leadless glaze, but the ordinary bath tubs which give a metallic sound when struck, are made of iron and covered with a lead glaze applied in the form of powder over the red hot surface. Wash-basins and kitchen sinks are enamelled in the same way.

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This is a large and important industry in the United States. In the 10 factories visited, which include the five largest, a little over 1000 men are employed in work which exposes them to lead. These factories are in Chicago; Sheboygan, Wis.; Louisville, Ky.; Chattanooga, Tenn.; Salem, Ohio; Trenton, N. J. and in Pittsburgh and three towns in the vicinity. The thousand men are employed in mixing, grinding and sifting enamel, and in applying it to the ware.

The cast-iron bath tubs, sinks and basins are first roughened by a sand blast and then placed by the enamellers in a furnace, heated red hot, brought out and sprinkled thickly with a powder which is essentially a ground glass containing soluble lead. Seven samples of enamel were subjected to the Thorpe test and were found to contain soluble lead in the following quantities: 0.51 per cent., 2.55 per cent., 6.31 per cent., 8.35 per cent., 9.04 per cent., 10.22 per cent. and 20.4 per cent. The lead used for making this enamel is red lead or litharge, and the mixture is always fritted, yet soluble lead is there.

The men who mix and grind are exposed to dust, the men who enamel are also exposed to dust and to great heat and to fatigue from the exertion of handling the heavy pieces. It is a very dangerous trade and the amount of lead poisoning is great, yet even in the best factories little is done to protect the men and in the worst, conditions are very bad. The enamellers are well paid, for it is work requiring skill, but 250 men whom I interviewed had averaged only 6 years in the trade. Mixers who are paid day laborers' wages are still more shifting.

Two hundred and seventeen cases of lead poisoning were found to have occurred among 1012 men during 1911, which would give a rate of 21.4 per 100 employed. This does not tell the whole story. It was excessively difficult to trace the cases in this trade, which employs so many non-English speaking Slavs but it so happened that I was able to make an intensive study of 148 men who were at the time out on strike and I found that one in three is probably the real proportion poisoned. Among these 148 men, 54 or 36 per cent., showed evidence of chronic plumbism and 38 others were not beyond suspicion. The form of lead poisoning among enamellers is often severe; 28 cases of palsy were found, and eight of the cerebral form of plumbism. Twelve were said to have been fatal, but only seven of these were reported by doctors. In Germany this is a leadless trade, the enamel used being lead-free and applied wet. In England it is leadless in some places, but in other factories a lead enamel is used and in these places the employers are obliged to follow strict rules for the protection of the men. In Austria where a similar method is used for enamelling stoves, there is, according to Teleky, a great deal of lead poisoning.

A comparison between foreign factories and our own is hard to make, but in a German pottery where glaze is made in the same way as enamel in our country no cases of lead poisoning has been reported among 15 mixers in 4

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years time, and in an English enamelling plant where a lead enamel is used, the average of lead poisoning among 26 employees is one in 2 years.

Lead Mining.—There has been no detailed study of lead poisoning among miners in this country, but in the course of an investigation of the smelting industry, one comes across cases in hospital records which show that the mining of lead ores is not devoid of risk of this sort. The Hospital of St. Vincent in Leadville has probably the oldest records of lead poisoning in miners and smelters in the United States. In 1880, the year after the opening of the hospital, 67 miners and 54 smelters were treated for this disease. The next year the miners numbered 41 and in 1882, 54, while the smelters rose to 119 and then to 424. For some years the cases among miners keep on at about 40 to 50, as long as the surface ores, the sulphate, carbonate and oxides, continue to form a large proportion of the output of the mines, but as the lower levels are reached and the ore contains largely lead sulphide, galena, the cases of lead poisoning fall off. Even in 1910, however, there were eight cases, but the next year only three and in 1912 all the lead poisoning came from the smelting plants.

It is commonly stated that lead sulphide ore is not dangerous, because the lead is in a form insoluble in human gastric juice. This has been held by eminent authorities to be true, on the basis of practical experience as well as animal experimentation. Rambousek stated at the International Congress of Industrial Hygiene in Milan, in 1906, that there had been no case of lead poisoning among 10,000 Bohemian miners while among 4000 smelters there were 150 cases. On the other hand, Biondi had seen such cases among the lead miners in Sardinia. The animal experiments performed by Blum confirm the theory of Rambousek as to the insolubility of lead sulphide, while those of Murgia and of Brezina and Eugling, seem to show that it is absorbed by the body.[10]

In the search for cases of lead poisoning among the smelters of the St. Louis region, I found 25 cases which had been treated in one St. Louis hospital, all of the men having come from the lead belt of southeastern Missouri, chiefly from Desloge. The cases were serious enough to require hospital care and one man had palsy, another a lead psychosis. They were foreigners and as there is but one small smelter in that region, employing only Americans, they must have come from the mines and the concentrating mills, where about a thousand foreign workmen are employed and where the ore is exclusively galena. To clear up the question as to possible poisoning from galena ore, Dr. A. J. Carlson, of the Physiological Department of Chicago University undertook to test the solubility of three of these Missouri ores in human gastric juice. He found the solubility to be: 1.38 per cent., 2.94 per cent., and 3.32 per cent., averaging about 2.5 per cent. There is, therefore, danger in mining lead ore even when the ore is pure sulphide.

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Lead Smelting and Refining.—The United States is one of the largest lead-producing countries and in addition to smelting and refining our own ores we refine much Mexican bullion. The 20 plants which formed the subject of a Federal investigation employ some 7500 men. They are situated in Perth Amboy and Newark, N. J.; Graselli and East Chicago, Ind.; South Chicago, Federal, Granite City, and Collinsville, Ill.; Herculaneum and Joplin, Mo.; Omaha, Neb.; Denver, Leadville, Pueblo and Salida, Col.; Murray, Midvale and Tooele, Utah, and East Helena, Mont.

The dangers in a smelting or refining plant are from fumes and dust and every man employed is more or less exposed to them, though in a clean, well managed plant there are parts which are almost free from danger. In a dusty neglected one there is not a single safe spot. In handling the ores, dust is the danger, though this varies according to the dampness of the ore and its composition, the sulphide ores being less poisonous than the carbonate and oxide ores. In pre-roasting, in roasting and in smelting there is danger from dust while the charges are being made up and put in the furnaces, and there is great danger from fumes while roasting and smelting proceed, and of both when the furnaces are emptied of their dusty and fuming product. In the later processes of refining the danger is chiefly from fumes.

The fact that these processes result in the evolution of lead fumes has led to the construction of elaborate flues and bag houses to catch and condense them. The fumes collect in these in the form of a very fine powder and the work of cleaning out and transporting this powder to the furnaces is very dangerous, except in one plant where it has been made possible to flush out the flues with water, and to empty the bag house mechanically.

The most dangerous places in an American smelter are: the open or Scotch hearths, where the ore is smelted in the open and the men exposed to fumes and to excessive heat and exertion; the emptying of the Huntington-Heberlein roasting pots, which in this country is always done in a way to cause much dust and fume; the charging of the blast furnaces, which in several plants has been made fairly safe but which is usually bad; the tapping of blast furnaces, and the cleaning out of flues and bag houses. Other furnace work may be equally bad if poorly managed but it is an easier task to prevent fumes in refining processes than in smelting. It is simply because of neglect that American refineries are on the whole worse than the smelters.

The provision of washing facilities for smelting employees is not nearly as important as the prevention of dust and fumes during work. Müller[11] has shown that the amount of lead that clings to a man's hands is insignificant in comparison to what he may be obliged to breathe in if the work is dusty and fumes escape. I have seen a plant with an elaborately equipped lavatory, bath house and dining room, whose rate of plumbism was nevertheless far higher than that of many plants with no washing facilities, because of the excessive amount of poisonous fumes in the open-hearth shed and of dust in the bag houses. If the lead on the hands were the great danger, there would

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be more plumbism among refiners and desilverers than among furnace men, for the former handle almost pure lead, but the fact is that the refining department is the safest of all.

We collected from physicians' records and from hospitals, 1667 cases of lead poisoning which occurred in 1 year, 1912, in 19 plants employing 7400 men. The twentieth had opened too recently to be included. This represents a rate of a little over 22 for every 100 employed. The British rate for that year was 1.8 per 100; the reports of four German smelters give a rate of 10 or 11 per 100, and in one large Austrian plant the rate was 9 per 100.

Manufacture of Storage Batteries or Electric Accumulators.[12]—As the use of motor cars and of electric lighting increases, the demand for storage batteries increases. There are a great many small plants in this country engaged in the manufacture or repair of these batteries and there are five large ones in which about 915 men are employed in work exposing them to lead. These are situated in Cleveland, in Niagara Falls, in Depew near Buffalo, and in Philadelphia.

The lead work consists in casting lead grids, trimming off the irregular borders, mixing oxides of lead with other ingredients, making a paste of the mixture, rubbing and pressing this paste into the grids, drying them, connecting them in pairs and groups by means of pure lead melted with an oxy-hydrogen flame, carrying them to the forming room where an electric current is passed through, and making the final connections with lead and the oxy-hydrogen flame. Fumes from melted lead and dust from lead oxides and from dried paste are the dangers here, to which must be added the danger that the poisonous oxides smeared over the men's hands may contaminate their food and tobacco. Personal cleanliness is of great importance in this work; yet only one of the five plants has complete arrangements for washing and bathing and all employ almost entirely foreigners who have little understanding of how to protect themselves. We found in the records of doctors and hospitals 164 cases of lead poisoning, chiefly acute, which had occurred in 1913 in the five plants employing 915 men, which means a rate of almost 18 per cent. This figure is incomplete, for in the case of two of the plants it was almost impossible to obtain any information. The rate in this industry in Great Britain was 3 per cent. in 1912. In a great factory in Germany in that same year the rate was 0.97 per cent.

The Painters' Trade.[13]—This is regarded in all countries as the most important of the lead industries, for it employs a large number of men and, as it is a skilled and well-paid trade, men do not drop out of it at the first attack of illness but usually keep on till actually incapacitated. It is harder to control conditions in the painting trade than in any other lead trade and therefore while industrial poisoning has been greatly diminished in other occupations, there has been no falling off in this trade in Germany or Great Britain.

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In the United States it is impossible to make any statement as to the amount of lead poisoning for which the painters' trade is responsible. All we can do is to gather scattered bits of information from various sources. The Illinois Report has 157 painters among 578 cases from all trades, or 27 per cent. Pratt's report of 109 cases includes no less than 42 painters. Andrews quotes the record of one New York hospital in which 59 cases of plumbism were treated, 28 of them painters, and the proportion among his 60 fatal cases was even greater, 40, or two-thirds. A local district council of the national trade union sent out a questionnaire to its members asking among other things as to a history of lead poisoning. Of 1009 who answered 185 gave a history of lead poisoning and 72 a history of kidney trouble, 77 of rheumatism, 27 of chronic stomach trouble.

In order to gain some idea of the extent of lead poisoning in the trade E. R. Hayhurst[14] made a thorough examination of 100 able bodied painters and found 59 of them with evidence of chronic plumbism. A hundred histories of lead poisoned painters showed a high proportion of severe cases, 39 per cent. of palsy, 9 per cent. of encephalopathy, 8 per cent. of general arteriosclerosis. The period of exposure before illness begins is longer in painters than in most lead trades. Only 19 out of 48 men had worked less than a year, 21 over 10 years.

The most dangerous branches of this industry in our country are: interior house painting, ship painting and certain parts of carriage painting, because these all involve the dry sandpapering of white lead paint, which dust-producing work is the most prolific source of lead poisoning among painters. The safest branches are the painting of farm implements, of farm wagons and of automobiles, when this is done on a large scale, for hand-work has given way to dipping by machinery and only the final decorating needs brush work. In smaller carriage and automobile shops and in the painting of railway coaches, hand-work persists and there may be much sandpapering, especially on the wheels, and in painting white milk carts, and the walls of toilet rooms in Pullman cars.

Ship painting involves the use of much red lead which must be sandpapered and must be chipped off when a new coat is to be put on. As this may have to be done in the small unventilated spaces between the inner and outer shells of the ship the amount of dust inhaled may give rise to severe poisoning. E. R. Stitt[15] of the U. S. Navy reported three cases of acute plumbism from this source. The men had been employed in chipping off old red lead paint from the inside of a ship and all suffered profound nervous symptoms, maniac depressive insanity in the first, epileptiform seizures in the second, and what appeared like an early stage of dementia præcox in the third. All recovered completely.

The prevention of lead poisoning among painters is fraught with much difficulty when the work is done outside a factory. House painting is often done in buildings where the water has not yet been turned on and there is

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no way for the men to wash properly before eating their lunch. In factories where this could be provided the arrangements are almost always quite inadequate. The dry sandpapering of lead paint is forbidden by law in several countries and it would be a simple matter to do away with it if the men could learn to use a cheap mineral oil to moisten the paper and catch the dust. The national trade union has already taken it up but many coach and carriage painters are outside the union and they need legal protection.

Manufacture of Rubber Goods.[16]—Lead, chiefly as litharge, but also as the basic sulphate, is used in compounding rubber. The basic carbonate and red lead are still occasionally found, but their use has been largely abandoned. The lead compounds are sifted, weighed and carried in open boxes together with crude rubber, sulphur and other ingredients, to mixing mills, where the box is emptied and warmed cylinders grind the materials together into a paste. As grinding proceeds a good deal of the dry portion falls off and is caught in a mill pan and the mixer is obliged to stoop down and with a long handled brush sweep up the powder and throw it back into the mill. This he does repeatedly for each batch of rubber and he may mix as many as 50 batches in a day.

The work of sifting, of weighing, and of mixing is dusty unless every precaution is taken in handling the lead and unless there is a good system of exhaust ventilation in connection with the sifting machines and the mills. Many mixing mills in American factories have been provided with hoods with suction to carry off the dust, but these hoods are usually poorly constructed and the exhaust seldom strong enough to be of any value. Cases of lead poisoning have been found to be not uncommon among rubber compounders and mixers in our factories, especially those in which the largest amount of lead is used, the footwear factories, plants for the manufacture of mechanical rubber, and tire works. White rubber and highly colored rubber cannot be compounded with lead, for the formation of lead sulphide blackens it and spoils the color. Hard rubber has little if any lead.

There has been no extensive study of lead poisoning in the typographical trades in this country nor in many of the industries in which metallic lead is used, nor in the grinding and making of paints and colors. Enough work has been done, however, to show that all the lead industries of the United States need regulation and that we can hardly hope to lower our high morbidity rate till we adopt the measures which have been followed with signal success in other countries.

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SECTION VI

MANGANESE POISONING

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Among the many forms of industrial poisonings from mineral and metallic substances, there are some which are infrequently seen on account of the restricted use of the substance or the rarity of its occurrence. Lead, arsenic and antimony occur not infrequently in the trades, and give poisoning forms which are easy of recognition. That there are others, has been known by workers in restricted fields for years, but on account of the small number of workmen who were subject to these forms of poisoning, they have been very little known by the medical profession at large.

Among these rarer poisonings we find as one of the most prominent that due to manganese and the manganese salts. Manganese is a metal belonging to the same group as iron, and like it forms both basic and acid salts. It is to some extent magnetic, which fact accounts for its name.

Toxicology.—The toxicology of manganese is very little known. In the first place the comparative rarity of the metal and the restriction of its use in the arts, and, in the second place, a still greater restriction in the field of medicine is responsible for this fact. In therapeutics we use manganese mostly in external applications in the form of potassium permanganate, and internally, to a much less extent, as manganese dioxide in the anæmias and some gynecological conditions. In commerce manganese is used most extensively as manganese dioxide in the manufacture of oxygen gas, while other salts, especially permanganates, have more or less use in certain trades.

Manganese poisoning is also seen in workers in industries dealing with other metals or their ores in which manganese exists as an impurity, and it was under such conditions that the author met his cases.

Schlockow in 1879 reported a peculiar disease among workers in a zinc mine in Upper Silesia, and the symptoms these patients exhibited were very similar to those later described by various authors in manganese cases. It is interesting to note that in the ore obtained from this mine there is a considerable quantity of manganese, and it also contained lead and arsenic in small amounts. Possibly some of the peculiar symptoms which Schlockow described in his cases may be due to the presence of lead and arsenic. Although he had no autopsies to confirm his opinion, Schlockow considered it to be a spinal cord disease which he ascribed to zinc.

More accurate observations were made by von Jaksch in 1901 in a factory

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where manganese dioxide and potassium permanganate were used. Von Jaksch ascribed the symptoms he saw to manganese and he described a clinical picture which corresponds to that later seen by Emden, Friedel and the author.

Symptomatology.—According to von Jaksch the principal symptoms are a gradual onset with impulsive laughter, weeping and mental alteration of many kinds. Later, gait disturbances develop, principally propulsion and retropulsion. Romberg sign is marked. The gait may or may not be spastic. The reflexes are exaggerated. Besides this he notes salivation, mask-like face and monotonous voice. Later the mental symptoms recede, and the condition tends to remain stationary.

Emden reported four cases of poisoning occurring in workers in a manganese dioxide grinding mill, where the atmosphere was heavy with manganese dioxide dust. His patients showed general muscular weakness with no atrophy, a mask-like face with inability to whistle. The gait was unsteady, and there was a tendency to fall forward on going down an incline. Retropulsion was marked. All tendon reflexes were active and equal. There was a coarse intention tremor which in some cases made writing difficult. The voice was unsteady and monotonous, and one patient had impulsive laughter. There were no sphincter troubles. He reported finding manganese in the urine of these patients, all of whom developed the symptoms after having worked a few months in the manganese dust.

The author has seen ten cases exhibiting neurological symptoms similar to those above in men who were working in the ore separating house of a large zinc mine, in which manganese occurs as an impurity in the ore to a considerable extent. In this mill the ore separation was brought about both by dry and wet processes. On the floors devoted to the dry separation the dust in the air was so thick that it resembled a heavy fog. The lower floor of the mill was devoted to the wet portion of the separation process, and was free from dust. All of the 10 cases occurred on the floors where the dust is thick, and, interestingly, most of them came from the lowest floor of the mill on which the dry separation was carried on, and hence where the dust was thickest. None of the patients came from the dustless floor where the separation under water is carried on.

Dust is undoubtedly the means by which the poison enters the system. Oliver and others have shown that most of our industrial poisons are taken in in the form of dust, not so much that which is inhaled as that which is swallowed in the saliva, or taken in with the food handled by the dusty hands of the workers. Von Jaksch reports that in the factory where his cases occurred no new poisonings developed when the dust was abolished. Emden ascribed his cases to the air of the mill thickly laden with the manganese dioxide dust.

All the author's 10 cases presented so many symptoms in common that there can be little doubt that they were due to a common cause; and they

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resemble so closely those described by other authors that one must in all fairness assume that manganese was the cause of the difficulty.

Other possible causes were considered in these cases. In the first place, zinc is the principal product of this mine, and exists in the ore in great preponderence over any other constituent. A careful study of the literature does not reveal any zinc poisoning with nervous symptoms which can in any way resemble those mentioned here. Popoff has described a case of general muscular atrophy with gastro-intestinal symptoms occurring in a bronze worker, which he ascribes to zinc. Of course it might as well, probably better, have been due to the copper, but in any event the cases here described bear no resemblance to this one. Edsall, after an extended experience among zinc workers believes that zinc never causes a systemic poisoning.

In another portion of the works connected with the mine where the author's cases occurred, where zinc occurs alone, and where the dust is not inconsiderable, no cases such as described here have ever occurred.

The possibility of lead playing a part in the etiology of this condition may be excluded when one considers that the zinc recovered from this mine is always of the first grade, and no measures are ever necessary to remove the lead. First grade of commercial zinc according to modern standards must assay less than 0.0025 per cent. of lead.

Another element of possible etiological importance which was considered here was the use of extremely strong electromagnets in the ore separation process. These magnets vary in strength between 20,000 and 100,000 ampere-turns. It is reasonable to exclude this as a causative factor for there are so many industries throughout the world where workmen are constantly moving in strong electric fields that were this to be a cause, the condition would undoubtedly be more commonly known that it is.

The symptoms and course of the disease shown in the author's cases is as follows: After the man has worked in the dust for a length of time varying from 6 months to 3 years he begins to notice that he has difficulty in walking, the main feature of which is that he is unable to walk down hill slowly. For years the foremen and shift bosses in this separator house have noticed that from time to time one of the workmen will become unsteady on his feet. This most often manifests itself in that when the workman would go down a slight incline with a wheelbarrow, and then attempt to stop suddenly he would fall forward into the barrow. This condition was so well known that the foreman on seeing this would say that the man was "zinced," and remove him to another part of the works, out of the dust.

On level ground and up hill the patient has no trouble at first, but when he starts to walk down an incline he goes faster and faster until at length he must run to keep himself from falling forward. He is unable to stop and must keep on with ever-increasing speed, until he runs against some object which stops him, or falls down. Retropulsion develops with this propulsion, and the patient finds himself unsteady on his feet whenever he wants to move.

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Walking backward is impossible, for as soon as the man tries it, he falls backward or sits down. This gait disturbance is very similar to that which is seen in paralysis agitans, and this is not the only way in which our cases resemble that disease. With the onset, pain and stiffness in the legs were noticed in four cases, insomnia in one and difficulty in writing in one. This latter patient complained that he could not control his hand well in writing, and that the figures he made were smaller than those he formerly made, a sign which occurs so characteristically in paralysis agitans.

While the symptoms of this condition are few, they are nevertheless very definite. The patient complains of little except propulsion, retropulsion and unsteady station. About half the patients have difficulty in hearing, not due to middle-ear disease. The speech is slightly slurring in some cases. The faces of all had the flat mask-like character of paralysis agitans, and there was in one a tendency to impulsive laughter. Tremor of the tongue is common as is also a fine, static tremor of the hands. The legs feel shaky and weak, and the station is insecure. Propulsion and retropulsion in gait are the most characteristic features. Asynergia is always present in well-developed cases. The eyes are normal in every respect. Muscular power is well preserved. The deep and superficial muscle reflexes are always normal. There is never any rest tremor of the limbs. Sensations are perfectly preserved. The laboratory offers little aid in the diagnosis. The blood never shows the changes so characteristically seen in lead poisoning. The urine is negative in those cases which have been investigated. The Wassermann reaction was negative in the five cases in which it was used, and the spinal fluid examined in three showed negative findings.

On the mental side nothing characteristic was found. One patient showed at first a definite intelligence deterioration similar to that of general paralysis, which later considerably improved. Still later he was subject to attacks of violence and temper, but these were brought on generally from some external irritative cause. Two patients complained of difficulty in memory and attention, which was not demonstrable objectively. Most of these patients came from a class with little or no education, which, combined with the language difficulty made routine mental examination extremely difficult.

Pathology.—Only one of the author's cases has come to autopsy, and here the pathological findings are so peculiar that one is reluctant to speak of them as of nosological importance, until further similar observations may confirm them.

The brain showed no gross changes, nor were any gross changes visible in the kidneys, liver or the spleen. On microscopic section the kidneys showed a moderate degree of chronic interstitial nephritis. In the liver there was considerable biliary cirrhosis present, and the liver cells themselves contained a great deal of pigment, part of which resembled lipochrome; while the majority of the granules were iron containing and stained densely with Prussian Blue reaction. Color reactions for manganese were not ob-

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tained in the liver or other organs. It is difficult to interpret the above findings. Such a large amount of iron in liver cells is rarely seen except in the severe anæmias where active blood destruction has occurred. There was no severe anæmia in this case, and the patient showed no other known toxic element than the manganese except alcohol, which he was said to have used to a considerable extent.

On section of the brain, no large tract system degenerations were seen. There was some degeneration of more or less regular character in the longitudinal fibers in the pons which run with those of the pyramidal tracts. While these degenerations were regular enough to be assembled into clearly defined tracts, nevertheless it was not possible to determine exactly either the upper or lower level of the tracts in question. The degenerated portion does not appear to go above the upper level of the pons, nor does the lower portion extend to any appreciable extent into the medulla. Here again we have a pathological change still more difficult to interpret than that mentioned above. In the first place, the descending fibers in the pons are not altogether well known. The pyramidal tract elements are clearly defined, and those of the fronto-pontine and temporo-pontine tracts are fairly so. Undoubtedly there are other fibers running in these bundles besides those mentioned, but what their origin and destination is it is impossible to say. In the degeneration seen by the author in this case, it is possible that we are dealing with descending degeneration of some fibers, only in the portion from the anterior border of the pons downward, as though the original focus of the degeneration had occurred in the upper pon or lower midbrain and the degeneration extended from there down. This is pure supposition, and we will need many more autopsies to prove this contention. Von Jaksch reported that he never found any pathological lesions post-mortem, but he does not state the details of his investigation.

Animal experimentation has so far offered no aid. Von Jaksch was unable to produce any symptoms in dogs who were forced to live in an atmosphere of manganese dust. The author has attempted, in a large series of rabbits, to produce a poisoning by mixing with the food of the animals the dust from the mine, the manganese containing ore, the pure zinc ore and pure manganese dioxide. Nothing was ever seen in any of these animals which could in any way correspond to the symptoms seen in the humans. Complete serial cross section of the brain of these rabbits has revealed nothing abnormal in the way of tract degeneration, or other pathological signs. Likewise sections of the liver in none of these animals showed iron or other pigmentation, as seen in the one human case. Perhaps it is not altogether surprising that symptoms and lesions should be lacking in these animals when one considers that the symptoms seen in the human are almost exclusively those of gait, of station and of facial expression. Such symptoms cannot be observed in four-footed animals, and if there are tracts governing the gait elements mentioned, one would not expect to find them developed in the least in the lower animals.

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It becomes obvious then that further investigation along the line of animal experimentation must be done with some of the higher forms, in which gait and station disturbances may be observed.

The course of this condition is very chronic. The prognosis for life is excellent, and for recovery poor. Generally the condition remains stationary, and rarely does it progress. Most of the cases show more or less regression of symptoms following their removal from the dust, but recovery has never been observed.

Treatment.—The treatment is also unfavorable, as in other forms of chronic metallic poisoning. Many authors speak of baths, of electricity and of massage and exercises as of value, but while there can be no doubt about the general value of such therapeutic procedure, nevertheless they do not seem to have any particular therapeutic excellence for manganese. Eliminative measures in these cases by means of the kidneys, the skin and the intestines are indicated, as in all cases of heavy metal poisoning.

In prophylaxis the greatest hope may be held out. There can be no doubt that the intake of the poison occurs by means of the dust, partly in inhalation, but more importantly in swallowing. The dust enters the gastro-intestinal canal together with the swallowed saliva, and with the food of the workers. Wherever it is possible completely to remove the air dust in mills, that is the best form of prophylaxis, for, as von Jaksch showed in the factory where he worked, no new cases occurred when the dust was abolished. Unfortunately, this is not always possible, but modern methods of blasts, suctions and ventilation appliances do a great deal to obviate this danger, and should be installed wherever the danger exists.

Personal cleanliness on the part of the workers is a very important factor. Properly equipped wash rooms, where the workers are obliged to wash and change their clothes before going to their homes will do a great deal toward obviating the bad effects. Possibly most important of all is the early recognition of the condition and the rapid removal of the worker from the dust-laden atmosphere.

But few cases of manganese poisoning occur among many workers, so here, as everywhere else, an element of personal susceptibility plays an important part in the etiology. To educate factory owners and managers where such symptoms are apt to occur is the most hopeful outlook for the prophylaxis, for it has been seen in the mill where the author's cases have occurred, that now that the condition is known, any man who shows symptoms even resembling those of manganese poisoning is immediately removed from the dust and set to work outside in the open. As a result of this, no more serious cases have occurred here in the past 3 years, and one or two individuals have been put to work outside on account of possible symptoms which are so slight that a trained observer could not with accuracy state that they were due to manganese.

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SECTION VII

MERCURY POISONING

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Translated from the German by LEWIS W. FETZER, M. D., Ph. D.

Symptomatology and Pathology.—In every case of poisoning the time at which a certain amount of poison is taken into the organism is of the greatest significance as to the kind of symptoms which follow.

This statement seems obvious when we compare acute and chronic alcohol poisoning because in these the differences are very readily apparent even to the layman. Therefore, we understand the term acute poisoning to mean poisoning that occurs in a limited space of time, say several hours, and as a result of taking one or more doses of a toxic substance.

When, however, we attempt to define the limits which bound the various states of poisoning, namely, the acute versus the chronic, we encounter difficulties. May we, for example, designate as chronic that kind of poisoning which follows the third, or even the sixth, application of blue ointment? Even though we recognize the existence of transitions from the acute to the chronic stages of poisoning, we are aware of the fact that under the designation of chronic poisoning we may have the greatest of variations. Only recently it was pointed out by T. M. Legge and Ellman, that in lead poisoning, which is the most frequently occurring form of industrial poisoning, the symptoms vary with the time at which the poison is taken. In mercurial poisoning the relation of the time of poison on intake to the clinical picture evinced is even more striking than in lead poisoning. In acute mercurial poisoning the clinical picture is governed by the manifestations in the intestinal tract. As an initial symptom we have profuse diarrhea in addition to the corrosion which may be present, but this is rarely observed in industrial cases of poisoning and is usually an accompaniment of poisoning from bichloride of mercury taken either by accident or with suicidal intent. The stools of acute poisoning are feculent at the beginning but soon become watery and profuse. In many instances according to Kunkle, the stools become bloody, sometimes contain pieces of mucous membrane and are of a very disagreeable odor. During the act of defecation a marked tenesmus and a severe pain in the lower part of the abdomen are present. In some cases vomiting occurs even when the poison has not been taken by way of mouth.

Simultaneous with the occurrences in the intestinal tract, renal symptoms become evident. These consist of a marked reduction in the output of urine, which may go on to a complete anuria. The urine in such cases contains, in addition to renal epithelium, white and red blood corpuscles. Con-

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sciousness is not affected but often the patients have headache and a feeling of fear, and near the end somnolence. With the loss in strength death usually occurs after several days.

When corrosive sublimate is taken instant pain and burning in the mouth occur as a result of the corrosive action of the substance, but where poisoning is effected by resorption of a solution of mercury through other channels, as the uterus or bladder, the major part of the symptoms in the buccal cavity which play an important rôle in subacute industrial poisoning, are absent.

I have never had the opportunity of seeing among the numerous cases of industrial poisoning which have come under my observation a marked case resembling the one described. Although I have been informed of the occurrence of slight diarrheas and despite the fact that I have the idiosyncrasy of becoming thus affected after washing in a solution of bichloride of mercury, I have never observed or read of a case of industrial mercury poisoning with pronounced acute symptoms. I have described acute mercurial poisoning simply because the probability exists that such cases may happen as a result of inhaling large amounts of mercurial vapors in establishments where the conditions are extremely bad.

In subacute mercurial poisoning the most prominent symptom is the inflammation of the buccal cavity. The patient complains of pain in the mouth and experiences difficulty in chewing at a time when other objective symptoms, save increased salivation, are lacking. Loosening of the gums soon follows, however, and they eventually swell, become red and bleed very easily. From under the reddened parts of the gums a thin pus exudes. The entire mucous membrane of the buccal cavity becomes involved in the process and on the mucous membranes of the cheek and lips sharply defined yellow ulcers develop, giving rise to a disagreeable odor from the mouth.

The stomatitis, with which is associated swollen lymph and salivary glands may reach a high degree of severity and result, in addition to the loosening of the gums and falling out of the teeth, in undermining the general health and, as the literature informs us, in a necrosis of the jaw bones. All degrees of stomatitis may result from industrial mercurial poisoning and there may be gradations from the slightest to the severest forms.

Mercurial stomatitis is not easy to differentiate from other varieties of stomatitis especially when other symptoms of mercurialism are lacking. It may, however, be differentially diagnosed from chronic gingivitis, a condition which affects workmen who take no care of their teeth, and one which is almost always found in older men. Chronic gingivitis lacks all the evidences of acute stimulation. The inflammatory process confines itself to the edges of the gums and does not extend to the remaining mucous membranes. Furthermore the profuse salivation present in mercurial poisoning is absent. In the lighter forms of stomatitis a purple red, slightly transparent line is evident which, however, is decidedly different from the lead line.

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In addition to the symptoms in the buccal cavity during mercurial poisoning, there may be minor manifestations noticeable in the digestive tract, even before the marked swelling, and pain in the mouth, and difficulty in eating are evident, such as a loss of appetite and slight diarrhea. With these manifestations, and often when all are lacking—the patient complains of indefinite pains in the extremities and joints. But above all things the patient shows evidences of psychic disturbances which are peculiar to mercury poisoning—Erethismus mercuralis. They consist of a peculiar form of stimulation and timidity and are evident more especially when the patient is in the presence of strangers. Nothing will describe this condition better than the statements made by the more intelligent class of patients, after cure. A technologist narrated the following to me.

"As a general thing I am a very energetic person and have taken part in many functions, but while affected with mercury poisoning I felt oppressed and uncomfortable, and was as shy as in my later boyhood days, in the presence of grown people."

A very intelligent workman, who had been engaged for many years in the experimental laboratory of one of the best known Austrian inventors, said:

"An invention had been completed, strangers came, and it was decided to demonstrate to them the manufacture of the respective commodity. As long as I was conscious of the fact that these people were standing behind I could not proceed to move a finger."

A French author relates an instance where a stranger came into an inn (or saloon) in which a number of hat makers, affected with mercurial poisoning, were drinking wine. During the presence of the stranger none of the hat makers was able to lift a glass to his mouth.

For a time these psychic manifestations were regarded as hysterical in nature, but this view has long since been dispelled.

The descriptions of manifestations just given are observable only in marked cases. This class of patients at the outset complain only of lassitude, tiredness and nervousness.

A further manifestation of the nervous system is the mercurial tremor. In marked cases of irritability or erethism one can often observe a slight twitching of the facial muscles, and a slight trembling of the fingers. The trembling, provided the patient continues to work with mercury, increases in intensity and finally extends to all parts of the body. In the early stages of the disease, and in lighter cases, only the hands and fingers tremble, but when the preliminary stage has passed, the tremors become of the characteristic type.

In addition to the fine tremors, which, however, are not always present, there may be interruptions by coarse contractions (jerks) and fluctuations. It is these coarse shaking movements which characterize the mercurial tremor. The mercurial tremor also goes through peculiar variations inas-

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much as it loses in intensity after ½ to 1 minute, and then picks up and becomes strong again. When the disease is progressive there is gradual successive involvement of the arm, the head, the vocal musculature, the legs and the trunk, and in consequence the entire body trembles and shakes. The patient loses more and more the control of the muscles, and the tremor present is of a pronounced intentional type. When at complete rest the patient does not tremble; this point is taken into consideration when differentiating the condition from paralysis agitations or the tremor of senility. Although persons affected with mercurial poisoning can perform heavy work, it is utterly impossible for them to do the kind which calls into play the finer muscular movements. This has been approximately expressed by an affected workman "the lighter the body the more difficulty I have in lifting it."

Cases are sometimes noted where persons affected with mercurial poisoning are still capable of carrying on a line of work to which they have become accustomed through years of practice, but are unable to lift a glass to the lips, and already have some difficulty in walking. Some patients drop to the ground on their way to work, and in consequence of the fact that each intentional movement will excite muscular contractions, are unable to rise unless aided by others. Even this class of patients, however, are able to work at their regular occupation in the factory.

Notwithstanding what has been said about these disturbances ataxia is never present, and it is only the shaking movements which make the desired movements impossible.

While drinking is difficult for patients with light forms of the disease those affected with heavy forms are absolutely helpless, and are unable to eat or clothe themselves, without aid. In the most severe cases the slightest movement in bed will excite contractions. Kussmaul to whom we are indebted for an excellent book on industrial mercury poisoning, describes cases of this severity. I personally never had a case of this kind under observation, as follows:

"The entire body is thrown to and fro and at the same time every group of muscles and each single muscle appear to be engaged in boundless activity. The head moves incessantly to and fro; at times it is jerked backward or sideward, the eyelids go up and down, and the eyeballs are rolled from one side to the other. The alæ of the nares and the mouth are jerked up and down, the face is contorted into all sorts of grimaces, the jaws move one upon the other, the arms and legs tremble and jerk together, and every individual muscle is engaged in the process.

The convulsions may become so severe that the combined strength of several strong men cannot control the jolts, which throw the patient to and fro, and it becomes necessary to restrain the patient in bed with strong straps to prevent him from being hurled out of bed."

In very light cases when nothing characteristic is present except coarse or fine tremors, this tremor cannot be differentiated from others, particularly

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from the alcoholic tremor. As the tremors become more intense and characteristic the jerking and shaking movements set in; the tremors may be sharply differentiated from the alcoholic tremor, the trembling of Basedows' disease and lead poisoning, and as previously stated from the shaking movements of paralysis agitation and the intentional tremor of senility. Difficulty in making a differential diagnosis can arise only when hysteria and multiple sclerosis come in question, but in these there are other nervous symptoms which will serve to determine their nature.

We have now discussed the cardinal symptoms of industrial mercurialism, viz. stomatitis, erethism, and tremors; the method in which these combine will be discussed later, as well as those which are less characteristic or occur seldom. Often when the trembling is of a high degree the extremities become weak. This fact has led the older authorities to speak of paralysis as one of the symptoms of mercurialism, although it must be said that cases of actual paralysis have been noted.

Letulle observed paralysis of the arms twice, and paresis of the legs once whereas I have noted radial paralysis in two patients affected with mercurialism in one of whom I saw it develop. Glibert noted as a general thing weakness of the hand and digital extensors in lead and mercury workers. As Leyden has reported mercurial polyneuritis after mercury inunctions and others (Ketli, Fawoeski) as a result of acute poisoning with corrosive sublimate, and as Heller has produced paralysis in animals by the injection of mercuric chlorid, mercurial polyneuritis undoubtedly occurs, but probably very seldom.

I have already remarked about the occurrence of a nephritis in these cases. On autopsy they show calcium incrustations in the kidney. The therapeutic administration of mercury, even when the mercury cure consists of giving small doses, may lead to an albuminuria and cylindruria (Lang and Welander) but, excluding the improper use of mercury, these conditions are transient. In the literature no clear accounts of nephritis in industrial mercurialism are given, but I have found albuminuria in a large number of patients with industrial mercurialism and, consequently, I feel justified in drawing the conclusion that the kidney is also affected in the chronic form of the disease. Mercury when administered cutaneously, subcutaneously, and by way of mouth for therapeutic purposes, often produce exanthema, and according to Ehrmann the mercury exanthema is one of the best known varieties of drug exanthemata.

Although the exanthema may be one of several varieties it is very often seen as a scarlatinous erythema which very frequently comes from the local application of mercury, as inunction with blue ointment. The erythema itches intensely and consists of punctate, closely crowded, reddened areas which gradually conflux to a livid surface, that has a higher temperature than the adjacent tissue. Finally desquamation occurs and scales are cast off in large lamellæ.

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Collis appears to be the only one who has observed cases with exanthema in industrial establishments but the case of a midwife poisoned as a result of washing in bichloride of mercury solution, reported by Wengler, might be included in this category.

Finally it may be mentioned that mercury workers show a peculiar copper colored redness in the trachea and the mucous membrane of the mouth; this is regarded by Kussmaul as a symptom of habitual mercurialism, when weakness, stimulation and tremor are also present. I have often noted this copper colored redness among workmen in the mercury works at Idria when other pathologic signs were absent.

Patients with the severe form of the disease have a striking paleness, lose their appetite and become emaciated. In the very worst cases the subjects are said to become cachectic.

The three principal symptoms of subacute and chronic mercurial poisoning, namely stomatitis, erethism and tremor, are not present simultaneously in all cases, nor in the same degree. We have cases which present no other symptom than a stomatitis and this is especially true of patients who take up large amounts of mercury in a relatively short time. This occurs in persons who do fire gilding. Stomatitis may therefore be regarded as a symptom of subacute poisoning.

On the other hand, we see patients in whom all evidence of stomatitis is absent, or in whom it was previously present, but who have the mercurial tremor in an accentuated degree. This form is found in hat makers and others who have imbibed very small amounts of mercury over a period of many years. I have seen only one case where a mercurial tremor, and here only a of light degree, followed several days of work with mercury. In cases of this character we probably have to do with persons who are especially susceptible to the effects of mercury. As a rule, it is necessary to take in small amounts of mercury continually for months or even years, or large amounts repeatedly at wide intervals. Erethism is not found in cases which develop quickly nor is it observable, save an intimation, in entirely chronic cases. In the latter the presence of erethism might be presumed if the tremor increases when the patient is conscious of the fact that he or she is under observation.

Erethism is most distinct in those cases which have the other two symptoms of mercurialism. There are undoubtedly cases in which the period of mercury intake was an average one, but just here we must not lose sight of the possibility that such cases might result by the imbibition of small amounts of mercury over a long period of time which would produce only tremors and then followed by the intake of large amounts of mercury in a short space of time which would yield an acute symptom as stomatitis. I will discuss the quantity of mercury necessary to produce poisoning in a subsequent paragraph.

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Receptiveness for Mercury.—From experience gained in the therapeutic use of mercury we have come to know that great differences exist among individuals in regard to receptiveness for mercury. Some have a distinct idiosyncrasy to react with it. Sachur cites the case of a 20-year-old girl who after an inunction with about 5 grams of blue ointment, containing 1.5 grams of mercury, developed numerous rhagades on the skin of the forearm and fingers and died with all the manifestations of an acute mercurial poisoning, diarrhea, bloody stools, and nephritis.

In the industries we also find distinct differences in regard to receptivity among the workmen. In these cases too the workers do not become affected at the same time. Where the conditions are not very bad only a part of the workmen become affected.

I have observed an instance of extraordinary receptivity in a strong 17-year old girl who became affected with mercurialism a short time after beginning work in a hat factory. The first indication was diarrhea, and this was followed some 4 months later by stomatitis, erethism and a gentle tremor. The girl, according to her statement, was principally employed in the dyeing department of the factory where she did not come into contact with mercury or mercurial compounds. Once daily, however, she was compelled to spend about 20 minutes in the felting division, where several cases of mercurialism had just occurred, handling mouth-wash water to workmen. The parents and grandparents of the girl although engaged in hat making and rabbit hair cutting (contractors) were never, as far as could be determined, affected with mercurialism.

I am unable to say on the basis of my own observations whether age or sex is a factor in mercurialism for the reason that men perform different kinds of work than women, and because the totally chronic form of the disease results from working years in the industry. However, if we use as a basis the knowledge gathered in therapeutics we must regard women and children, and tuberculous, anæmic, and cachectic individuals as most receptive.

The amount of mercury necessary to cause poisoning is said by Göthlin to be 0.4–1.0 mg. taken daily over a period of several months; this was determined by laboratory tests.

Tests made by Dr. R. Lang, at my suggestion, showed the dust from the blowing department of a hat factory to contain 2.338 per cent. of mercury. Granted that the air in an establishment of this kind will carry 40 mg. of dust per cubic meter, the amount of mercury inhaled by an individual would be 5.561 mg. per working day. Only 0.0576 mg. of this, however, is soluble in the digestive juices.

Mercury on the hands of workmen and workwomen may also gain entrance into the body. In this connection we have the observation of Heim who collected 4 mg. of mercury from the hands of a scrap cutteress, but only 0.5286 mg. of it was soluble.

As only a small part of the mercury on the hands gets into the mouth

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it is very evident that under the most unfavorable conditions a workwoman engaged in the blowing room of a hat factory will take in from this source, and by inhalation, a combined amount of mercury which is less than the figures reported by Göthlin.

When we take cognizance of the fact that Göthlin observed cases of poisoning in which mercury was taken over a period of months, and furthermore that the author has noted only cases in the subjects employed in the one relatively well-equipped factory for over 10 years, we must conclude that the smallest amount of mercury taken continuously over a long period of time will eventually produce manifestations of the disease.

This statement, however, must be amplified by mentioning the fact that in all probability only that portion of the mercury which is resorbed by the body, and not that present in the digestive organs or the mercury which has gained entrance to the body by other channels, is detrimental. Notwithstanding this, and leaving out of consideration the entirely different conditions of resorption in the lung, we must not lose sight of the fact that resorption of mercury does occur from the stomach, although the amount is small, and furthermore that some mercury is rendered soluble during the process of intestinal digestion.

The investigation conducted by R. Lang in 1912, which I touched upon above, yielded the following data with rabbit's hair which had been treated with a mercurial mordant.

Unblown mixed hair, digested with 0.346 per cent. HCl yielded	0.018 per cent. sol. mercury
then digested with 0.346 per cent. HCl + 0.1 per cent. pepsin	0.044 per cent. sol. mercury
then digested with 0.2 sol. of Na_2CO_3	0.025 per cent. sol. mercury
then digested with 0.2 sol. of Na_2CO_3 + pankreon	0.018 per cent. sol. mercury.
	0.105 per cent.

From these data it is evident that by progressive digestion the amount of mercury rendered soluble is greater than by gastric juice digestion alone.

We are appraised of a similar behavior for lead by Legge and Goadby (Lead Poisoning and Lead Absorption, London, 1912).

Göthlin has determined that when the amount of mercury inhaled does not exceed 1 mg. per 4 cubic meters of air, practically all of it is resorbed.

From what has been said in the foregoing it is obvious that the method whereby mercury enters the organism and the kind of mercury compound introduced, is of the greatest significance in mercury poisoning.

In industrial mercurial poisoning, however, chiefly the element mercury (metallic and gaseous) and mercuric nitrate come into consideration; mercuric chloride and mercuric cyanide are only occasionally etiological factors. The point as to which of the portions inhaled, that gaining entrance to the stomach with swallowed saliva or the part taken up by the lungs, is the most dangerous must be left undecided for the present.

Accordingly the greatest danger to those who have to do with mercury

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lies in the fact that mercury will volatilize even at ordinary temperature. In this connection Renk was able to show that at a temperature of 10°C. 1 cubic meter of air above an area of ½ square meter of mercury would contain the following amounts of mercury:

5 cm. above	1.86 mg.
50 cm. above	1.26 mg.
1 meter above	0.85 mg.

The volatility of mercury increases with a rise in temperature, which is shown by the following table taken from Kunkels' toxicology, 1901, page 133:

GRAINS OF MERCURY IN A CUBIC METER OF AIR ACCORDING TO VARIOUS INVESTIGATORS

Temperature	Regnault	Hagen	Hertz
0°C.	0.236	0.177	0.00224
10°C.	0.305	0.205	0.00569
20°C.	0.409	0.231	0.01430
30°C.	0.563	0.276	0.03083
40°C.	0.785	0.338	0.07787

The elimination of mercury occurs briefly in the lowermost portions of the intestine from where it is discharged with the feces, and also in the urine, saliva and sweat. The elimination from all channels is slow and not continuous.

Oberlander found mercury in the urine 190 days after giving the substance in a mercury cure. The elimination in this 190 days' period, however, was not continuous for there were often spaces of time, 10 days, in which no mercury would be noted in the urine. In the examination of organs of subjects succumbing to acute poisoning the largest amounts of mercury were found in the kidney, liver and large intestine.

Treatment.—In the treatment of industrial mercurial poisoning it is obvious that the first thing necessary is to keep the patient from all work which involves the slightest possibility of taking up mercury. The next thing necessary is to bring about the elimination of the mercury—how far this is possible we are not able to say—by stimulating intestinal activity, the secretion of urine and secretion of sweat (sweat baths). The administration of sodium or potassium iodide seems to be indicated in these cases.

Above all things these patients must be strengthened by giving good food, fresh air, and hydrotherapeutic treatment. The stomatitis is treated symptomatically with gargles and painting with astringent fluids.

The stomatitis when appropriately treated is usually cured in the course of a few weeks and in light cases in a few days. The discharge of the erethism requires a somewhat longer period of time, but it also vanishes completely when the patient is kept from mercury work.

The removal of the tremor in cases of average degree of severity usually

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requires several months but in young patients it can be discharged completely. In individuals around and over 50 years of age the prognosis is not always quite so favorable. I have seen persons who had ceased working with mercury still show a marked tremor 5, 6, 15 and 30 years afterward. Although I have repeatedly seen marked improvements and even cures in older patients, it is always advisable to refrain from forecasting a favorable prognosis in these cases. In the older literature many cases are described in which the mercurial tremor and the stomatitis are said to have led to a fatal result. Mercurialism should most often be regarded as an indirect cause of death inasmuch as it lowers the vitality of the organism and thus prepares the soil for a tuberculous infection. With regard to the effect which mercurialism will have upon the progeny it is often stated that mercurial poisoning will produce abortion or will tend to bring forth weak offspring. There are in fact cases described in the older literature which support this contention. No proof exists, however, to show that mercurialism in the male has any detrimental influence upon the progeny.

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SECTION VIII

NAPHTHA AND BENZOL POISONING

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A study of the literature of poisoning from the products of petroleum such as naphtha, benzine, gasoline, and from the products of coal-tar distillation, such as benzol, reveals a great deal of confusion and the student will often be left with a question in his mind as to just which one of the many complex bodies belonging to these two groups was involved in any specific instance of poisoning. This is partly because in the majority of cases a mixture was used, not a chemically pure substance, but it is also true that to distinguish between the physiological effects of poisoning by benzine and by benzol is difficult if not impossible. The similarity of names adds to the confusion. Benzene is a synonym for benzol, not for benzine, but it is not always possible to be sure, especially in French and Italian reports, exactly what body is meant when one of these words is used. Another confusing factor is the similarity of uses of the two groups of chemicals. Though it is clear that the fumes in a benzol still or in a petroleum refinery are of fairly well-known nature, it is not possible to say which will be present in a rubber factory, a dry-cleaning establishment, a varnish factory, or a room in which motor cars are tested, for both benzine and benzol are used for all these purposes. Therefore it is easy to see why some observers have not attempted to decide the real nature of the poison which was present, while others have reported their cases in such a way as to arouse doubts in the minds of critics.

V. Jaksch,[1] Roth,[2] Lewin,[3] Rambousek,[4] emphasize the differences between the petroleum distillates and the coal-tar, holding that the latter have a special toxic action on the central nervous system. Lehmann,[5] whose exhaustive article is the most authoritative pronouncement we have on the question, finds a difference in degree between the toxicity of benzol and benzine, but not a difference in kind. It seems, however, best to treat the two classes of substances separately, at the same time realizing that in some instances it is not possible to discover just what compound was involved.

Petroleum and Its Distillates.—Petroleum is a dark brown, oily liquid derived from the subterranean distillation of vegetable matter. It has a structure of enormous complexity, differing in different regions. Pennsylvania petroleum contains a complete series of hydrocarbons from CH_4 to $C_{16}H_{34}$, also the solid hydrocarbons $C_{35}H_{52}$, $C_{27}H_{56}$ and $C_{30}H_{62}$. When distilled, the lighter compounds pass over at 30°C. to 170°C. These are the

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benzines, naphthas and gasolines and contain 60 per cent. to 75 per cent. pentane (C_5H_{12}, b. pt. 36.3°C.) according to the boiling point. The compounds which distill at 30°C. to 70°C. are used chiefly for motor cars; those at 80° to 120°C. for extracting fats, as cleaning fluids, and, in combination with the distillates at temperatures up to 170°C., in varnish and as turpentine substitute in paint.[6] Next in order of distillation comes kerosene, illuminating oil, and then the solid paraffines, vaseline, etc. Different proportions of these bodies are found in petroleum of different regions. West Virginia oil is said to have a good deal more of the heavier bodies than Ohio oil and Russian oil is heavier than American.

Lehmann[5] and his colleagues experimented with different benzines, letting animals inhale the fumes, and they found the effect to be irritation of the respiratory mucosa, muscular twitchings and a slowly increasing narcosis. Light benzine in large doses was decidedly less poisonous than the heavier. With Gundermann, he found that inhaling 20 mg. of light benzine had little or no effect, but when as much as 45 mg. was used they experienced a feeling of impaired mentality with weakening of the will power. Much larger doses were used by Felix[7] in experiments carried out on prisoners in Bucharest. He administered benzine as one would chloroform for anæsthesia. While some individuals proved to be quite insusceptible, even to large doses, the majority after inhaling 5 to 15 grams for 7 to 12 minutes felt dizziness, nausea, smarting of the conjunctiva, and in some cases burning in the chest and drowsiness. Larger doses caused sleep and anæsthesia, succeeded by nausea, vomiting, headache, dizziness, depression and drowsiness.

There are several records in the literature of poisoning after drinking benzine, but industrial benzine poisoning always takes place through the inhalation of fumes or through skin absorption, although so far as is known the latter gives rise to local symptoms only.

The effects of acute poisoning from the inhaling of moderate amounts of fume resemble mild alcoholic intoxication, and in dry-cleaning establishments or rubber works, where the employees are very familiar with the condition, they speak of it as "naphtha jag." Dufour[9] speaks of the excited gayety of girls employed in poorly ventilated dry-cleaning establishments, a gayety soon yielding to depression, headache, clouded mentality. In the spring of 1913 I had the opportunity, through the courtesy of Dr. J. H. Landis, Health Officer of Cincinnati, to interview nine interior house painters who had recently experienced the effects of using a quick-drying paint, containing large quantities of benzine, in small and practically unventilated rooms. Dizziness, headache, spots before the eyes, dryness and choking in the throat, burning of the eyelids, were complained of by all, while some complained also of nausea, vomiting, pains in various parts of the abdomen, and painful urination. Several said that at the end of the day they could no longer do fine work and two had attacks of momentary blindness. One suffered from ulcers of the lips and gums. In several instances the worst discomfort came

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on leaving work, the dizziness and staggering coming on in the open air. Loss of appetite and a feeling in the morning of having been drunk the night before were usual features of this mild benzine poisoning.

More serious symptoms follow exposure to heavier fumes. Lewin[8] says that in the oil regions of Pennsylvania and in the refineries at Point Breeze he was told of men who when they went down into the tanks became so excited and irrational that they could with difficulty be persuaded to come out. In still more serious cases, which are rare in proportion to the numbers employed, there is a rapidly increasing weakness, quick and weak pulse, labored respiration, delirium and coma. A physician in a town where there is a large rubber factory told me of such a case in his practice. The patient, a strong man, had been dipping wooden forms in a tank filled with a solution of rubber in benzine, to make seamless surgeons' gloves. He felt dizzy and ill and left work, but on his way home he staggered and would have fallen, had not two men helped him. Later, when in bed, he lapsed into unconsciousness and when the physician saw him he was comatose, very pale and almost pulseless. He recovered completely.

Wichern[11] describes an accident in a benzine tank where some remnants of benzine were floating on the surface of the water poured in to clean it. A man who climbed in was overcome by fumes and fell with his face in the benzine. He was quickly rescued but was unconscious, his muscles strongly contracted. He had severe chills and vomiting and though he recovered he had some irritation of the throat for 2 weeks.

Cleaning out vats and tank cars is one of the most common causes of acute benzine poisoning.[4] The latest report of the German Factory Inspectors[12] tells of a workman who entered the benzine cylinder of a washing machine in a dry-cleaning establishment while there was still some benzine left in it, and was found lying unconscious overcome by the fumes, although the machine stood by an open door. Fumes from the benzine used in quick drying paints were responsible for two cases of poisoning noted in the last report of the British Factory Insepctors.[13]

Another industry which is an increasingly important source of benzine poisoning is the automobile. Gowers[14] has reported a very interesting case of pseudomyasthenia, involving chiefly the muscles of the throat and tongue, in a tester of motor cars who was continually exposed to fumes of gasoline in a state of incomplete combustion. In our own country Potts[15] has described a case of "encephalitis" due to gasoline poisoning in a man who worked for 4 months filling automobile tanks. After about 2 months of headache, nausea, and visual disturbances, he fell unconscious while at work and when aroused complained of intense headache. There was weakness of the right third nerve and of the left side with partial incoördination of the left arm and the gait was cerebellar in type. The weakness of eye and arm were permanent.

A very extensive instance of gasoline poisoning occurred in the digging of

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a tunnel in Montreal and involved no less than 42 cases. Fumes from the gasoline engine were responsible.

Fatal cases are rare. Lehmann[5] has collected several from the literature and added one, a man who spilled 40 liters of benzine in a low cellar, tried to scoop it up, came out complaining of feeling faint and tired and a half hour later was found lying dead outside the door. Zimmermann's[16] fatal case died while trying to remove with benzine the crusts which had formed on the inside of a tank. Lewin[3] heard of fatal cases of poisoning in the American oil fields and refineries among the workmen who have to enter the tanks. One of the English cases of poisoning from the use of benzine in paint ended fatally. These paints are especially dangerous when the surface to be painted is warm.

A striking case of fatal poisoning by petroleum products is given by Korschenewski,[17] who has had long experience in the Russian oil regions. A healthy young man had worked all day at the pumps which control the flow of naphtha from a well. At evening he felt weak and ill. The next morning he expectorated quantities of tarry blood which did not redden on exposure to the air. By noon he was jaundiced, there were purpuric spots in the skin, vomiting of blood, delirium, convulsions, and death on the following day. As we shall see later this resembles closely some of the cases of fatal poisoning from benzol.

Chronic benzine poisoning is said by many to be rare, the workers in rubber factories, dye-houses, etc., having no more ill health than falls to the lot of all factory people. Lehmann[5] and Goldschmidt[18] say that German benzine workers show little if any effect from the fumes and Lewin[8] found the ordinary work in American oil wells and refineries to be attended with no noticeable effect on health. On the other hand Russian writers, who have had experience in establishments where conditions are bad, find much ill health traceable to the constant inhaling of benzine fumes. Berthenson[19] reviews the literature on this subject and shows that in workers who are employed for long periods there develops bronchial catarrh, dyspepsia, malnutrition, anæmia, nervous derangements (Korschenewsky, Petkewitch). In the Baku oil region among 8465 employees there were, in 1895, 1475 cases of respiratory disease, chiefly bronchial catarrh, and 1216 cases of skin disease.

Dorendorff's[20] cases are usually quoted as striking instances of chronic benzine poisoning. The men were rubber workers and the most prominent symptoms complained of were pains of a tearing character in muscles and joints, tenderness on pressure, weakness and coldness of the right hand, psychic depression, loss of memory, difficult speech. There was increased excitability of the skin to mechanical irritants and heightened tendon reflexes.

In addition to poisoning from fumes there is a toxic action exerted on the skin by the petroleum products, especially the heavier. Paraffin workers develop multiple papillomata, or pemphigus-like or eczematous eruptions.

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Benzine also causes skin lesions. Last year many cases of skin diseases were reported from the printing trades of Berlin,[21] and were traced to the introduction of a new, benzine-containing fluid for cleaning ink from type. The disease was described as a localized eczema, with redness, tenseness, blisters and later scaling.

More often the lesions caused by contact of the skin with petroleum are described as typical acne lesions. Lewin[8] found this in the American oil country. The irritating effect of the oil causes inflammation of the sebaceous ducts, retention of secretion, suppuration, multiple skin abscesses, etc. In the great factories for agricultural implements in this country the men and girls employed in the preparing and spinning rooms in the twine mills often suffer from very distressing acne. This twine is treated with a mixture of fuel oil 95 per cent. and wool oil 5 per cent. to soften it and keep it from being devoured by field insects when it is in use. The workers feel the effects of this oil most in summer when they work with their sleeves rolled up because of the heat.

Benzol.—Pure benzol is C_6H_6 and boils at 80.4°C., but the commercial variety contains substitution products as well, chiefly toluol and xylol, distilling at 80° to 85°C. It is a solvent for rubber, fats, gums, celluloid, and therefore much used in industry, but not as much in this country as benzine. Commercial benzol contains also a trace of carbon bisulphide, and there is a variety used in Germany in the rubber trade and for varnish which has 16 per cent. or even 60 per cent. of this compound.

Lehmann[8] and his colleagues and Chassevent and Garnier[22] have come to the same conclusions as the result of animal experiments, namely, that the symptoms caused by crude, by commercial and by pure benzol are the same in kind but that the action of the first two is more rapid and intense because of the presence of toluol and xylol, both of which are more toxic than benzol. Rambousek,[4] however, finds pure benzol more poisonous than the two others and so do Agasse-Lafont and Heim.[23]

Symptoms.—The symptoms caused by the inhalation of these fumes point to a poison with a selective action on the central nervous system, and they are as follows in the order of their appearance: muscular tremors, salivation, violent twitchings, exhaustion, paralysis, narcosis, respirations first quick then slow, rapid pulse, low temperature. Usually the animal recovers but cats may show a striking idiosyncrasy and die after a short exposure, from respiratory failure. Lehmann[5] found no characteristic changes in organs or blood but Chassevent and Garnier[22] found congestion of the abdominal organs and peritoneum, and ecchymoses and ulcers along the upper border of the stomach in the mucous coat. Santesson,[24] experimenting with crude and with pure benzol, was able to produce hemorrhages into the mucous membrane of the stomach and the intestines and into the lungs. He concludes that benzol dissolves the body fat and that emboli of fat lodge in the smaller vessels causing first stasis and then rupture of the vessel wall. Selling[25] found blood

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changes in rabbits as a result of poisoning with pure and with commercial benzol, the most striking feature being a leucopenia which progressed till the white corpuscles numbered 20 in a cubic millimeter of blood. He thinks benzol a specific poison for the leucocytes.* In human beings, benzol is decidedly more toxic than benzine. Lehmann and Gundermann[5] produced symptoms in themselves with 10 grams of benzol which required 45 grams of benzine to produce. They consider it a poison of only moderate severity, while Rambousek[4] finds the vapors of benzol mixed with air very poisonous and publishes a large number of serious and of fatal cases collected from the literature and from personal information.

The majority of serious cases of benzol poisoning have occurred in work which exposed the man to very heavy fumes, usually in the cleaning out of some receptacle in which benzol had been kept and which in some cases still had remnants left when the cleaning began. Other men were at work with benzol stills where from some accident or neglect fumes were allowed to escape. Slower forms have been observed in rubber works, dyeing and cleaning establishments, in a cannery where a solution of rubber was used instead of solder. The fatal case reported by the factory inspectors in Great Britain in 1912[13] was employed in a benzol still, the two German cases in this same year[12] were employed, one in a still, the other in using coal-tar paint, and the two Austrian cases,[27] also fatal, were in rubber works.

Coal-tar paint is coming into use increasingly in recent years as an anti-rust paint for iron and steel. When used in the open air as in bridge painting, not much discomfort is experienced by the painters but when it is applied hot and fuming in an enclosed space there may be actual poisoning from it. Last year when inspecting the paint department of one of the great ship yards I found a paint in use consisting of Trinidad asphalt in coal tar. This was applied hot to the tanks and the water bottoms, a term used for the space between the inner and outer shells of the hull. Dense white fumes escaped from the hot mixture which were irritating and nauseous to me at a distance of 15 ft. and in the open air, but the men seem to establish a certain tolerance to it. Certain ones, however, are more susceptible and I was told of instances of semi-intoxication, coming on either during work or on reaching the open air, and one case of wild delirium, which subsided into confusion and bewilderment when the man was brought to the fresh air.[10]

The descriptions of the serious cases of benzol poisoning, when given in detail, vary a good deal, yet all present the picture of intoxication by a substance with a specific action on the central nervous system. In addition, there are a number of instances of lesions of the blood-vessels leading to multiple hemorrhages. The Austrian factory inspectors report in 1911[27]

*Other blood changes noted are (Simonin[26]) a transient eosinophilia of 25 per cent., and (Dorendorff[20]) a brown pigment in the plasma and leucocytes.

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two fatal cases of an unusual illness in rubber workers, characterized by purpuric spots on the body and found to be caused by benzol fumes.

Lenoir and Claude[23] tell of a fatal case of purpura in a workman employed for several years in a dye house and exposed daily to fumes of benzene. There were subcutaneous hemorrhages, bleeding from nose and from gums, increasing cachexia and sudden death. At autopsy, bloody effusion into the pleural cavity, hemorrhages into the mucosa of stomach and intestines and under the endocardium, myocardial infarcts.

Santesson[24] has described the most famous cases in the literature of benzol poisoning. Nine young women were employed for periods of a few weeks or months in a velocipede tire factory in Upsala. For a while before the appearance of the most serious symptoms they had been working overtime, 12 hours or more a day. The symptoms complained of were usually those already described as characteristic of acute benzol poisoning, but the more serious cases had in addition purpuric spots on the skin and a tendency to hemorrhage, sometimes excessive, from nose, mouth or gums. In one there was a menstrual hemorrhage. Four girls died, after illnesses lasting a month or two. Profound anæmia developed, in the third case reaching 600,000 reds, and the fourth case had a striking leucopenia, hardly any whites being discoverable in a cubic millimeter of blood. Santesson found that the solution used in the factory was a crude coal-tar benzol distilling at 80° to 90°C.

Lehmann[5] thinks that the severity of these cases, so much greater than any reported in the literature and with the unusual feature of purpuric lesions, is to be explained on the ground of the youth and sex of the victims and the overexertion to which they had been exposed. Three almost precisely similar American cases reported by Selling[25] seem to have escaped Lehmann's attention. These also were young girls, younger than the Swedish cases for they were only 14 years old, and it is this immaturity which probably explains the more rapid course of the disease in Selling's cases. The girls were employed in a cannery near Baltimore where recently a solution of rubber and rosin in commercial benzol had been substituted for solder in the sealing of cans. The work was done in a well-ventilated room and there is no note of excessive hours, but it was during the summer months when the heat in Maryland is intense. Three girls were brought to the Johns Hopkins Hospital suffering from anæmia, purpuric spots, dizziness, hemorrhages from throat, nose and gums. As in Santesson's cases, the symptoms increased after the exciting cause had ceased and two of the three girls died in a few days with symptoms of profound toxæmia. There was a count of only 1,090,000 reds, falling before death to 640,000, and the hemoglobin fell to 8 per cent., but leucopenia was the most striking feature of the blood picture. The whites numbered only 480 per cu. mm. and the loss affected chiefly the polymorphonuclears, which formed 16 per cent., the small

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mononuclears forming 71 per cent. and the large, 10 per cent.* Selling calls the condition one of aplastic anæmia, for in spite of the great loss no regenerating forms of blood corpuscles were found. He found the substance to be crude benzol chiefly, and with it as also with pure benzol he was able to bring about in rabbits a marked leucopenia, reaching 20 per cu. mm. at which point death occurred.

It is worth noting that in Selling's cases and in two of Santesson's there was fever, while in experimental benzol poisoning a fall of temperature seems to be invariable.

Preventive Measures.—The prevention of poisoning from fumes of benzol or benzine lies in good ventilation so that the fumes may be diluted down to the point of harmlessness. Even allowing for an individual susceptibility such as we have reason to think does exist in some people, the really serious cases of poisoning have all been caused by exposure to very heavy fumes, and the reports of chronic poisoning come from those countries chiefly where industrial hygiene is comparatively neglected. High ceilings, abundant ventilation, scrupulous cleanliness are enough in cases where fumes are not heavy; artificial ventilation with air exhaust must be installed when the danger is greater, preferably, according to the British inspectors, with a down-draft as these are heavy fumes. If a vat or tank must be cleaned out, the men must be furnished with divers' helmets. For the distressing skin diseases nothing but very strict bodily cleanliness will help and in susceptible cases there is no hope of cure as long as the work is kept up.

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* The resemblance of these symptoms to those of scurvy is striking, especially as v. Jaksch found leucopenia in scurvy.

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SECTION IX

CHRONIC PHOSPHORUS POISONING

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Industrial phosphorus poisoning is always chronic. It may occur in chemical works where phosphorus is obtained from mineral phosphates, or in the manufacture of matches from white phosphorus, or in the use of phosphor bronze, or through exposure to the fumes from ferrosilicon, or possibly through the use of acetylene gas in which phosphorus is contained as an impurity.

In recovering phosphorus from mineral phosphates, the process is carried on under cover to hold in the fumes, and the phosphorus collected and kept under water. The work is, therefore, almost free from danger, yet there are a few records of cases of phosphorus necrosis in this industry. Pieraccini[1] says that the preparation of phosphorus from calcined bones in Italy is attended with danger of necrosis. The British report[2] on phosphorus poisoning states that in the one large English factory where phosphorus is prepared from apatite there have been 17 cases scattered over a period of 50 years. In France[3] nine have been reported from the chemical works since 1880.

Ferrosilicon is used in the manufacture of steel. It is an alloy of iron and silicon, the latter being present in proportions of 15 to 95 per cent. and containing arsenic and phosphorus as impurities. In the presence of moisture ferrosilicon which has less than 30 per cent. or more than 70 per cent. of silicon decomposes, giving off arsiniuretted and phosphuretted hydrogen. Fatal poisoning has occurred from these fumes,[4] chiefly on ships which were carrying a cargo of ferrosilicon and in which steerage passengers were housed in ill-ventilated cabins near the hold. Such cases cannot be regarded as of industrial origin, but they serve as a warning that the use of ferrosilicon in industry is not free from danger.

Phosphor bronze is an alloy of copper, tin, and a small amount of phosphorus, which should not exceed 2 or 3 per cent. Kaup[5] has found cases of phosphorus necrosis in Austria where phosphor bronze is used in the making of cannon and of cartridges. His attention was first called to this danger by a case which occurred in 1897, and he found that the man had been working with an alloy consisting of copper 90 per cent., tin 8.9 per cent., and phosphorus 0.76 per cent. From so small a quantity as this, phosphuretted hydrogen was given off in amount sufficient to cause poisoning.

Another source of phosphorus poisoning in the United States was recently uncovered in New Jersey. Miss Lilian Erskine, of the New Jersey Depart-

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ment of Labor, is authority for the statement that in the making of fireworks in that state a paste of white phosphorus may be used and in such a way as to expose the men and girls employed in certain departments to the fumes of phosphorus and to direct contact with the paste. The method is said to have been brought over to this country from Italy.

A possible source of phosphorus poisoning, according to v. Jaksch,[6] is acetylene gas which contains this element as an impurity, and, according to Hölzer,[7] the use of phosphorus in the making of miners' lamps.

All these industries are of little importance compared with the making of phosphorus matches, which is the phosphorus trade par excellence, employing as it does many more work-people than all the others put together. The first description we have of industrial phosphorus poisoning was written 12 years after the invention of lucifer matches. Lorinser,[8] an Austrian physician, wrote in 1845 concerning 22 cases of necrosis which had occurred in the match factories near Vienna. The first recorded case in America is probably to be found in the books of the Massachusetts General Hospital in Boston for 1851. This was a man 48 years of age, who for 14 years had been employed in paste making and in dipping matches. He had no symptoms pointing to phosphorus poisoning till 18 months before his admission to the hospital, when he had had pain in one of the lower incisors and soon after lost the tooth. The record describes a stage of necrosis already far advanced, with swollen lower jaw, bloated face, eyes dull and staring, breath very offensive and half a dozen fistulous openings along the lower border of the jaw. The upper jaw was also affected. Later entries show extension of the disease and increasing toxæmia ending in death some 6 months after his first admission. Lorinser's work was followed during the next few years by publications on the subject of phosphorus necrosis in Germany, France and England, and from that time up to the present day no year has passed without a large number of reports as to the prevalence and the prevention of this disease.

It is the most dreaded of all the industrial diseases, not because it is the most dangerous, for the mortality is only 15 to 20 per cent., nor because it is widespread, for in this respect it does not compare with lead poisoning, nor because it is sure in its action, for in reality only a small minority of those exposed are ever affected, but because of the great and prolonged suffering caused by the local disease and the shocking deformities which almost always result. The mere fact that phosphorus attacks the bones of the face and makes its effects visible in pitiable disfigurement has probably had much to do with the vigorous efforts which have been made to do away with this industrial disease.

In the making of matches three kinds of phosphorus may be used, but the only one which concerns us is the white or yellow, the amorphous and the sesquisulphide not being dangerous. Workers in white phosphorus match factories make the paste, dip the sticks in the paste, carry the matches to

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drying rooms, carry them from drying to packing rooms, and pack them in boxes. All these processes are attended with danger from the fumes of phosphorus, especially the first two.

According to T. E. Thorpe,[2] phosphorus exposed to air at ordinary temperature volatilizes, forming fumes which consist of phosphorous oxide, about 5.8 per cent.; phosphoric oxide, 73.1 per cent.; and phosphorus, 21 per cent. Thorpe found that these fumes have a solvent action on human teeth, for carious teeth exposed to phosphorus for 12 hours lose 0.37 per cent. of their weight. Fumes dissolved in the saliva also have a solvent action on the teeth. Dilute phosphoric acid was allowed to flow over fragments of broken teeth for 3 hours and at the end of this time 8.9 per cent. of their weight was lost.

Thorpe's analysis of samples of air in a match factory showed that at a dipping table covered with phosphorus paste there was 0.02 mg. of phosphorus per 100 liters of air, and in the middle of the boxing rooms, 0.12 mg. per 100 liters. An analysis of the water in which the workers had washed their hands after 4 hours' work showed an average of 4.2 mg. per head for a day of 10 hours.

Pathology and Symptomatology.—Industrial phosphorus poisoning is a chronic, local disease, the so-called phosphorus necrosis or "phossy jaw." Usually it has its starting place in a carious tooth, or perhaps in a root exposed by receding gums. It is essentially a periostitis of the alveolar process of one or the other maxillary bones, resulting in the formation of abscesses, sloughing of the periosteum and necrosis of the denuded bone.

The first symptom is a toothache, increasing in severity. If it is neglected pus forms under the periosteum and before it finds an outlet the pain is excruciating. Extraction of the tooth lays bare a wound filled with offensive pus, and it is characteristic of this form of poisoning that the extraction wound should be very slow to close. If before healing is complete the patient goes back to work, the opening does not heal and the inflammation spreads, the periosteum is loosened and sloughs off and the exposed bone undergoes necrosis. If the lower jaw is the one affected an active proliferation of periosteum and formation of callus goes on along the margins of the destructive process, and the jaw looks thick and prominent, but in the upper jaw there is little effort at repair. The necrosed bone comes away from the upper jaw in fragments, while in the lower a sequestrum forms which is held fast by the callus and is not spontaneously cast off.

The primary effect of phosphorus poisoning is a change in the periosteum making it less resistant to infection; the secondary effect is the entrance of pyogenic organisms through a cavity or a defect in the mucous membrane of the mouth and the setting up of suppuration, first in the alveolar periosteum and in the gums, later in the surrounding tissues. The late symptoms of phosphorus poisoning are chiefly the symptoms of suppurative disease.

As the pus which has formed at the root of the tooth burrows its way to

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the surface and discharges, there is relief from the severest pain, but the fistulous openings thus formed are very slow to heal and may continue to discharge pus for months or even years. If the disease is in the upper jaw the fistulæ tend to pass down into the mouth, but when it is in the lower, the pus works its way down and breaks through the cheek or neck. There may be ulceration of the lining of the nose. Röpke[9] found in a factory in Solingen 19 out of 64 workmen with ulceration of the nostrils, and in two perforation of the septum had already taken place.

In the majority of cases healing takes place after a year or two with the loss of several teeth and a part of the alveolar process, but in other cases the destruction is much more extensive, leading to loss of large portions or even the whole of the lower or upper jaw bone. In the worst cases both upper and lower jaws must be removed. Andrews[10] reports such a case from an American factory, the man being able to take only liquid food afterward till his death 22 years later. Suppuration may extend to the bones of the face and cranium, or to the orbit, destroying the eye, as in one of Andrews' cases and in two reported by Teleky.[11] When the disease is in the upper jaw there is danger of extension to the meninges, an accident which always ends in death.

Phosphorus poisoning is typically slow in onset. Oliver's[12] cases had worked from 7 to 15 years when necrosis developed. Lewy's[13] 82 cases averaged 6.6 years' exposure; Hirt's 87, 5 years.[14] Teleky[11] gives his record as follows: 3 employed less than 1 year; 47 from 1 to 5 years; 41 from 6 to 10 years; 41 from 11 to 20 years, and 13 more than 20 years. Yet there are some unusually rapid cases, especially among youthful workers. Lorinser[8] had one which followed 7 weeks' exposure. Garman,[15] who was for many years physician to a large English match factory, saw nine fatal cases, all dying at an early age: two were 19 years old, one was 21, three were 22, one was 23, and two were 27.

The lower jaw is said to be affected in about 60 per cent. of all cases. Teleky[11] and Hirt both found the proportion to be five of the lower to three of the upper. Villaret,[16] however, found the upper oftener involved and so did Kuipers.[17] Garman[15] found the numbers practically equal.

Apparently sex does not play any part in influencing susceptibility to phosphorus poisoning. The majority of cases in recent years have been among men, because the most dangerous work, mixing and applying the paste, was done by men, while the women had the safer work of packing. Teleky[11] had but 83 cases among women to 139 among men, though there are four or five times as many women as men in the Austrian factories.

It is a disease of long duration, lasting, under treatment, usually from 1 to 2 years but often much longer, and when recovery does take place it is rarely complete, for the new bone is not sufficient for real repair and the sinking in of the soft parts, together with the scars from healed abscesses and fistulæ, result in terribly disfiguring deformities.

The mortality under modern treatment is from 15 per cent. to 20 per cent.

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When death occurs early in the course of the disease, it is from sepsis or meningitis; when later, it may be from the cachexia of chronic sepsis or from tuberculosis. Chronic digestive disturbances contribute to lowering of the vitality and are due, at least in part, to the continual discharge of pus into the mouth in cases with prolonged suppuration.

Individual susceptibility is an important factor in this form of poisoning, for only a very small minority of those exposed ever develop necrosis, even of those with carious teeth. The same thing is found to be true of animals exposed to phosphorus fumes; only a few succumb. No absolutely complete statistics are to be found as to the occurrence of phosphorus poisoning among match workers, but the following are fairly so: Hirt[14] reported 70 cases during 20 years among 600 workmen in the Silesian factories; Magitot[18] gives nearly the same figures for the French factories, 70 in 21 years among 620 workers. The British report of 1896[2] states that among 1701 persons employed in occupations exposing them to the fumes of phosphorus were 300 men who, during the preceding 5 years, had had 16 cases of necrosis, and 1401 women who had had 14 cases, making 30 cases in 5 years among 1701 persons.

There has long been a controversy as to whether phosphorus poisoning is to be regarded as a purely local or a systemic disease. The earlier writers, especially the French, considered it a general poison invading all the tissues and manifesting itself, even in the absence of necrosis, by general cachexia, anæmia, respiratory disease, dyspepsia, albuminuria, and even paralysis. To this condition Magitot[18] gave the name "phosphorisme," and he believed that the phosphorus worker may keep on for years without evidences of poisoning so long as his functional equilibrium is maintained, but if there be an injury to the mouth, such as the drawing of a tooth, the disease will become active. He therefore advised an expectant treatment as long as possible. Pieraccini[1] thinks that though necrosis is rare, it is a mistake to conclude that therefore phosphorus poisoning is rare, for general phosphorisme may exist without necrosis.

On the other hand, the physicians appointed by the French Government[3] to inquire into the health of match workers found no evidence of general ill health. Mahu[19] says that among tobacco workers 7 out of 11 deaths are caused by tuberculosis, but among match workers only 5 among 15. The British report to the House of Lords[2] states that there is little if any phosphorisme among these people in England, and Oliver has seen necrosis in strong and otherwise healthy people. According to Helbig,[20] the German factory inspectors usually report the general health of match makers to be good.

That phosphorus has a specific action upon the bony system in general is, however, not denied. Long exposure to the fumes of phosphorus seems to set up changes in the periosteum of other bones than those of the jaws, making it more vulnerable, less resistant to infection. The most significant

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case of this kind is Wegner's[21] reported in 1872. A man of 19 years had recently left the match factory in which he had worked since he was 4 years old without any symptoms of poisoning either local or general. He was injured by the wheel of a wagon passing over his leg and received two flesh wounds not extending to the bone. Gangrene set in necessitating an amputation above the knee-joint, during which it was discovered that the periosteum of the femur was loosened and dead bone protruded from the wound. The destruction of periosteum increased, extending up the thigh, and the man died of sepsis on the sixth day. Wegner found at autopsy a slight general hyperostosis of the cranial bones, an ossifying periostitis of the alveolar processes of both jaws, and bony deposits on the epiphyses of the long bones.

The experiments which Wegner made on animals showed that under the influence of phosphorus fumes there occurs a thickening of the subperiosteal bone at the expense of the Haversian canals, narrowing them and thus partially shutting off the vascular supply to the bone. Layers of very compact tissue are laid down in the region of the epiphyseal cartilages. In the jaw bones there is an enormous thickening of the periosteum and so much swelling of the soft parts that the animal can no longer open its mouth. Necrosis with softening and sloughing is found only when there has been an injury to the mucous membrane leading down to the bone. The same fact was established by the earlier experiments of v. Bibra and Geist.[7] It is only exposure to fumes that sets up these changes in animals; feeding them phosphorus has no such effect.

Twenty years after Wegner, Riche[28] discussed these experiments and pointed out the bearing which later studies in bacteriology have had on our conception of phosphorus necrosis. The osseous changes, Riche said, were attributable to phosphorus, but the necrosis was caused by the action of microörganisms on this changed tissue. Phosphorus produces changes in all the bones, but necrosis takes place in the bones of the jaw because they, more than any, are exposed to the entrance of pus-forming bacteria. Teleky[22] also emphasizes the importance of the secondary effect of pyogenic organisms in the case of phosphorus necrosis and believes that a careful examination of the histories of patients with so-called phosphorisme would show that the cachexia was due to chronic sepsis or to tuberculosis.

The effect of phosphorus on the periosteum in lowering its resistance to infection is seen in those cases of which there are several in the literature in which the necrosis developed long after the person had left the atmosphere of phosphorus fumes. In the French factory at Trélaze[3] the use of white phosphorus was discontinued in 1894, but 3 years later a pronounced case of necrosis developed. V. Bibra and Geist[7] reported the case of a 23-year-old girl who worked a few years in a match factory and felt no ill effects except an occasional toothache. A year after she left the factory she was seen by the authors and was then in an indescribably terrible condition, dying of sepsis.

Another evidence of alteration in the bones as a result of phosphorus

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fumes is found in an abnormal fragility of the long bones which fracture as a result of a slight injury or even of muscular action only. This "fragilitas ossium" has been reported by several French writers, by Brocoorens in Belgium,[2] by Kocher in Berne,[8] by Teleky in Austria,[11] by Garman[18] in England, and by Andrews[10] in America.

Treatment.—It is still a matter of controversy whether the treatment of necrosis should be early and radical or expectant and conservative. Some dentists advise the extraction of all carious teeth if the person has been working in a match factory; others prefer to fill those that are in the first and second stages of caries. In either case there is no question that the patient must be forbidden to return to the factory till the mouth is in a sound condition. When the pulp is exposed, extraction is imperative and the resulting wound must receive careful treatment till it is healed. Abscesses must be freely incised, cleaned and drained. When necrosis of the bone has taken place there are many who advise immediate subperiosteal resection through healthy bone. Kocher[2] follows this plan and claims 83.7 per cent. of cures. Kuipers[17] has had 89.0 per cent. by the same method, while Garman[15] waits for a sequestrum to form before he operates and claims that he has 83.0 per cent. cured. Teleky[11] has lately come out for the expectant treatment. He believes that the early resection of large parts of the jaw has seemed to give good results largely because the relapses have not come under the observation of the surgeon. In order to bring about healing in such an early operation, it is necessary to remove a much larger portion of bone than if one waits for the formation of a sequestrum. This does prolong the disease, but results in less deformity and less disturbance of function. Therefore unless the disease is too protracted or the danger of sepsis too great he advises against early extensive operation.

Preventive Measures.—The prevention of phosphorus necrosis has been shown to depend absolutely on the prevention of the manufacture of white phosphorus matches. The history of the match industry in Europe, in Great Britain and in our own country shows that years of preventive work through the removal of fumes, the substitution of machinery for hand work, and the medical and dental care for the workers have been unsuccessful, for cases of necrosis have continued to appear even in the best-managed factories. Phosphorus necrosis does not disappear so long as white phosphorus is used in industry. France has had the longest experience in the making of non-poisonous matches and Leclerc de Pulligny[24] reports that since 1898, when the use of white phosphorus was finally given up in all the State factories, the special precautions which were formerly used have also been given up, but no case of phosphorus necrosis has occurred.

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SECTION X

THE PREVENTION OF PHOSPHORUS POISONING

BY JOHN B. ANDREWS, Ph. D., New York City, N. Y.

Phosphorus poisoning arises in the extraction of phosphorus, in the production of phosphorus compounds, such as igniting agents, in the extraction of coal-tar dyes, in the making of phosphor-bronze, and in the manufacture of matches. In America, as elsewhere, the last-named industry has been the main source of the disease.

White or yellow phosphorus was discovered in 1669 by Brand, of Hamburg, while he was experimenting for a liquid to transform silver into gold. It is now obtained commercially by treating bone ash with sulphuric acid, filtering and evaporating the product, heating this with charcoal, and afterward distilling it. It is, when pure, a colorless, transparent, crystalline substance, which readily becomes yellow upon exposure to light. It is volatile at ordinary air temperature, and in addition to its poisonous qualities is highly inflammable.

The ignition of phosphorus by friction was discovered in 1680, but although the first friction match was invented in England by John Walker in 1827, it was not until 1833 or thereabouts that phosphorus was utilized in the composition of match heads, in Vienna. In 1836 the first American patent for a friction match was granted to Alonzo Phillips, of Springfield, Massachusetts, and in 1842 Reuben Partridge invented the first splint-cutting machine. From that time to the present the principal inventions of machines in this industry have been made by Americans.

Three kinds of phosphorus are now used in match manufacture, to which correspond three types of matches.

One is the "safety match," which must be struck on a prepared surface. This match contains no phosphorus and is harmless. The igniting composition is painted on the box and contains the red or amorphous phosphorus discovered by Schrotter in 1845, which is made by baking the poisonous white or yellow variety in a closed vessel, and is consequently more expensive. The red phosphorus is insoluble in the human digestive tract, and when pure is therefore non-poisonous. Although used almost exclusively in one or two countries of Europe, the manufacture of "safety" matches is, in America, still very limited.

The second kind of match—the familiar old-style "strike anywhere" parlor match—is now happily fast disappearing. The match can be struck on any ordinary rough surface, but possesses the disadvantage that the paste

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used for the heads contains from 5 to 7 per cent. of poisonous phosphorus, the other ingredients being glue, powdered glass, oxide of zinc, chlorate of potassium, and some coloring mixture. To the presence of the poisonous phosphorus can be traced the phosphorus necrosis among workers in match factories. The use of this poisonous element in match manufacture is now prohibited in the leading countries of the world, including the United States where this result was accomplished through the imposition of a prohibitive tax. An older form of the phosphorus match is the sulphur or card match, so named on account of the sulphur in the head and the card form in which the matches were sold. Due to the disagreeableness of the sulphur fumes this match has fallen into almost complete disuse.

The third variety of match also possesses the desirable quality of striking anywhere and is at the same time non-poisonous. This is the strike anywhere match now manufactured and used in those countries where public sentiment has been sufficiently aroused to prohibit the use of white phosphorus. In France, where the substitute for white phosphorus was discovered and where it was first used, the match business has been a government monopoly for more than 25 years, and as such threw upon the government the burden of the human loss resulting from the peculiar hazards of the industry. Government officials noticed that the profit they had hoped to receive from the business was rapidly drawn away in the form of compensation for sickness and death from phosphorus poisoning. Experts were set to work and in 1898 Sevene and Cohen found a substitute for the poisonous element, in the sesquisulphide of phosphorus. This substitute has not only been successfully employed in France but has been introduced almost everywhere where white phosphorus is forbidden.

Although complicated by modern methods and machines, the fundamental processes in the manufacture of matches may be described in a few words: The wooden match splint is prepared; the phosphorus composition for the head of the match is mixed; one end of the splint is dipped into the paste; the "green" match is allowed to dry; and finally it is boxed and wrapped.

The lumber from which match splints are made is usually poplar or basswood. White pine makes the best splints, but it is hard to get. Blocks which have been selected so as to be free from knots and of a certain dimension are fed into the machine by an operative called a "block feeder." The machinery holds the block against knives, which cut out the splints and force them into perforations in a series of hinged iron plates which move as an endless chain over the entire length of the machine. Projecting from the lower surfaces of the iron plates, the splints move from the front end of the machine into a bath of melted paraffine. They are then passed over a roller which is coated with phosphorus paste by running in a trough containing the composition, which trough is in turn supplied by a pipe leading from a covered tank on the machine holding a supply properly heated. This last operation puts the heads on the matches. The finished matches are

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now ready to be dried, which is accomplished by the movement of the endless chain of plates over a series of drums, bringing them during their transit in contact with a number of air currents. The time for drying varies with the weather, but usually takes an hour. Arriving again at the front of the machine, the matches are mechanically punched out of the plates, and are then handled by the filling mechanism, passing finally to a revolving table at which girls are seated who slide the covers on the boxes.

If the poisonous variety of phosphorus is used, practically every process in this series following the preparation of the splints is dangerous. In the mixing, dipping, drying and packing rooms the danger from breathing phosphorus fumes, although it may be somewhat diminished by thorough ventilation and by the rigid enforcement of preventive measures, is always present. Particles of phosphorus also become attached to the hands of the employees and are later transferred to their mouths.

For over three-quarters of a century the dangers incident to the use of white phosphorus in this industry have been known. As early as 1838 Doctor Lorinser, of Vienna, diagnosed in a female match worker named Marie Jankovits the first authentically recorded case of industrial phosphorus poisoning. The rapid reporting of additional cases led the Austrian government to appoint a committee of inquiry, but the committee's recommendations apparently never became effective, for in the 10 years 1866 to 1875 there were reported 126 cases of phosphorus necrosis in the hospitals of Vienna alone.

The death of a match maker in London about 1896 and the consequent newspaper discussion of unhealthful conditions in the trade fixed British attention upon industrial phosphorus poisoning. A total of 105 cases, 19 of which resulted fatally, were reported in Great Britain up to the end of 1900, but the record is admittedly incomplete. While in Great Britain less than 1 per cent. of match makers were found to have suffered from phosphorus necrosis, in Switzerland it was formerly 1.6 to 3 per cent., and in France 2 to 3 per cent.

In America a surgeon published as early as 1855 a pamphlet giving the history of nine serious cases of phosphorus poisoning in New York City. One of these earliest cases required the removal of an entire jaw.

In 1864, 12 more serious cases from these same match factories were recorded in a letter to the New York Evening Post. At that time there were 75 match factories scattered throughout the eastern states, but the toll taken by phosphorus from these many establishments is unknown. That phosphorus necrosis, however, was a common malady 25 years ago among match factory employees in this country is not denied by anyone.

In the course of their general investigation into the conditions of women and child wage-earners in 1908 and 1909, agents of the United States Bureau of Labor found 16 definite cases of phosphorus poisoning among match factory operatives, and learned that many other cases had occurred, not only

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many years before, but also in recent years and in some of the factories where conditions were the best. This discovery, and the related activities in European countries, led to an intensive study under the joint auspices of the Bureau and of the American Association for Labor Legislation, of 15 of the 16 match factories then in operation, all of which were using poisonous phosphorus.*

The 15 factories investigated employed, according to statements by the manufacturers, 3591 persons, of whom 2024 were men, and 1253 were women 16 years of age and over. Children under 16 numbered 314, 121 boys and 193 girls. Detailed investigation showed that 65 per cent. of the employees were working under conditions which exposed them to the dangers of poisoning. The women and children were much more exposed than the men, 95 per cent. of the women and 83 per cent. of the children under 16 years of age being so exposed.

An investigation by the writer in the homes of the work-people of three of these factories yielded a total of 82 cases of phosphorus poisoning. In two factories at least eight perfectly authenticated cases were found to have occurred in 1 year, and references were obtained to three more. In one small factory records were secured of more than 20 serious cases during the past 30 years, many of them requiring the removal of an entire jaw. In one of the most modern establishments, records of 40 cases were secured. Of this number 15 resulted in permanent deformity through the loss of one or both jaws, and several cases resulted in death. In another factory the records of 21 cases were secured, six of which were in the year of the investigation. In all, the records of over 150 cases, of which four were fatal, were collected in a very short time, although it had been the claim of the match manufacturers, and there was a popular impression to the effect, that the trouble had not existed in this country in a serious form for 20 years.

Preventive Measures.—The early manufacture of matches was often carried on in dirty and ill-ventilated workrooms which exposed the employees to every danger of phosphorus poisoning. With the increasing demand for matches conditions grew worse until within 11 years after the phosphorus match was discovered, government investigators in Europe were studying the conditions which brought on the disease. The infant industry was driven out of the cellars. The rules issued by the various governments in the attempt to "regulate" the effect of the poison upon the health of workers included such provisions as closed-in machinery, exhaust and ventilating systems, separate lunch rooms, dressing rooms and wash rooms, with mouth wash and soap and towels furnished by the employer, medical and dental inspection at the expense of the employer, and factories built according to architectural plans furnished by the government. The hours of labor were also strictly regulated, and women and children were rigidly excluded from the most dangerous depart-

*Phosphorus Poisoning in the Match Industry, by John B. Andrews, Bulletin 86, United States Bureau of Labor.

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ments. The percentage of phosphorus in the paste was limited until many manufacturers rebelled and made matches in defiance of the laws. Notices warning employees of the dangers involved in the handling of phosphorus were posted, and employers were required to read the warnings to their employees at stated intervals. Partly as a result of these extraordinary and expensive precautions the industry was rapidly concentrated in the hands of a comparatively few employers and conditions were improved, but wherever special study was made phosphorus poisoning was nevertheless found to exist in a serious form. During all of the past generation, in spite of attempts on the part of humane employers and governments to regulate the evil, the malady continued to claim its victims one by one. So serious was the situation that the regulative method of dealing with the problem gradually became recognized as totally inadequate.

In 1872 Finland gave up attempts at regulation and enacted the first law prohibiting the use of white phosphorus in match factories. Denmark, in 1874, with a similar experience, did the same. For 40 years they have had no cases of phosphorus necrosis. France prohibited the poisonous ingredient in 1897, Switzerland in 1898, and the Netherlands in 1901. In 1906, on account of the difficulties of eliminating the use of phosphorus in countries with an important export trade, the International Association for Labor Legislation secured an international conference at Berne which resulted in an international convention providing for the absolute prohibition of the manufacture, importation or sale of white phosphorus matches. The convention was signed by Denmark, France, Germany, Italy, Luxemburg, the Netherlands, and Switzerland, to which were added Great Britain in 1908 and Spain in 1909, together with numerous colonies of the signatory states. Without signing the convention, Austria enacted a prohibitory law in 1909, New Zealand and New South Wales did so in 1910, Hungary in 1911, Mexico in 1912 and Canada in 1914.

In the United States, which was prevented by constitutional limitations from signing the convention and where the national government may not regulate directly the conditions of industry within the states, resort was had to the federal taxing power. By a law enacted by Congress in 1912, to go into effect on July 1, 1913, a prohibitive tax of 2 cts. a hundred was levied on matches made of white phosphorus. Their importation and exportation were also absolutely forbidden after January 1, 1913, and January 1, 1914, respectively. Heavy penalties, ranging up to $5000, or imprisonment for 3 years, or both, were provided for violation of the act.

Thus throughout practically the whole civilized world, before the close of 1914, the use of poisonous phosphorus in the manufacture of matches has been definitely prohibited. And with such prohibition there was abolished from the match industry that most loathsome of occupational diseases, phosphorus necrosis.

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CHAPTER II

OCCUPATIONAL INFECTIOUS DISEASES

SECTION I

ANTHRAX

BY LANGDON FROTHINGHAM, M. D. V., Boston, Mass.

Synonyms.—English: anthrax, splenic fever; in the human species, malignant pustule, wool sorter's disease, rag-picker's disease. French: Charbon, Fièbre charbonneuse, Charbon bactéridien. Italian: Antrace, Febbre carbonchiosa. German: Milzbrand, Milzfieber, Karbunkelkrankheit.

The Bacillus anthracis is intimately associated with the history of bacterial infection. Owing to its large size, ease of cultivation, rapidity of growth, and the facility with which it may be stained, it perhaps has been and is, more frequently employed than any other organism for morphological and biological study. It was first seen microscopically by Pollender in 1849 in the blood of a cow that had died of anthrax. It was later observed by others and in 1863 Davaine showed it to be the cause of the disease. It was therefore the first bacterium to be definitely associated etiologically with an infectious disease of animals. Its artificial cultivation by Koch (1876) and Pasteur (1877) established a new line of thought and study and Pasteur's classical experiments in attenuation and protective inoculation laid the basis of subsequent theories of immunity. Therefore it has been well claimed that the *Bacillus anthracis* is the very "cornerstone" of modern bacteriology.

Morphology.—According to various observers the anthrax bacillus varies in length 1.5 to 10 μ and in width from 1.0 to 2.5 μ. It is non-motile, multiplies by direct division and under certain conditions by spore formation. For example, "spores never develop in the tissue fluids of the living animal, in the unopened carcass, or inside of the meat of slaughtered animals" (Hutyra and Marek), but they invariably appear when the organism is cultivated on artificial media and are also formed in nature under proper conditions of heat and moisture. The spore occurs as a relatively large oval body situated in the center of the bacillus. The bacilli usually appear in shorter or longer chains. In the blood, organs, and local lesions, such chains are composed of two to a dozen or more bacilli, while in culture media the chains are frequently so long that the number of individual bacilli cannot be estimated. The ends of the bacilli are square though sometimes, according to some writers, rounded or even slightly bulbous and occasionally even slightly concave. In the blood of animals, each organism and chain of organisms is surrounded by a distinct capsule readily brought out by certain

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staining methods. Cultivated upon artificial culture media—with the exception of fluid blood serum—this capsule does not appear. Degeneration or involution forms of the bacilli are not infrequently met with in preparations of blood or tissue pulp—especially if putrefactive changes have occurred—as well as in culture media. In these forms the protoplasm stains irregularly or not at all, just the outer shell being visible and this may be more or less filled with dark staining granules. Sometimes also capsules are observed which are either empty or contain remains of bacilli.

The bacillus is readily stained by any of the simple stains and is Gram positive. The spores may be stained by the usual methods recommended to color these bodies.

Cultural Characteristics.—The bacillus is a strict aërobe though Rosenthal by depriving it more and more of oxygen succeeded in converting it into a facultative anaërobe. Development occurs on artificial media at any temperature between 12°C. and 43°C. but the optimum is about 35°C. and at this temperature an extensive growth is evident in 12 hours. Spores are said to form at temperatures between 18° and 42°C.

On some of our laboratory media the growth has certain characteristics which, however, are not diagnostic (although at one time so considered) for a number of bacteria belonging to this group possess very similar characteristics and confusion is easy even if such organisms are cultivated side by side with anthrax. Thus the "Medusa" colony on the gelatine plate, the inverted fir-tree in the gelatine stab (with rapid liquefaction of the media in both instances); a somewhat similar colony on the agar plate and agar slant; the gradual liquefaction of Loeffler's blood serum; the clear bouillon (without pellicle) above a cotton-like sediment (best seen by lightly agitating) which soon becomes more like sputum—such characteristics must be considered of limited diagnostic value only, not alone on account of the group characteristics referred to, but also because of deviation from type due to slight variations in different lots of culture media, different "strains" of anthrax, and different conditions of cultivation.

Twelve- to 24-hour cultures on plain agar slants made for diagnostic purposes from the blood, organs, or local lesions of animal or man, are frequently contaminated with other organisms especially if made from animals several hours after death. If such cultures show colonies—and sometimes there may be only one—or a more diffuse growth somewhat resembling morphine sulphate crystals or damp snow, with perhaps flaring edges, and further appear (with the hand lens and transmitted light) like frosted or even corrugated glass, suspicion is at once aroused and proof of anthrax must be established by animal inoculation (guinea-pig, or better, mouse).

Tenacity.—The vegetative rods are readily destroyed by heat and chemicals, the spores on the contrary are very difficult to kill. Thus the bacilli may be killed by a dry temperature of 55° to 58°C. in 10 to 15 minutes, while dry heat at 140°C. for 3 hours is necessary to destroy the spores. Moist heat, on

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the other hand, as steam at 95°C., will kill spores in 10 minutes and steam at 100°C. in 1 to 3 minutes, and boiling water will kill spores in 5 minutes. Low temperature, even −100°C. for an hour, does not injure either bacillus or spore, and at −180°C. (liquid air) spores and bacilli are still alive after 3 to 15 hours (Ravanel, Belli). Bacilli will survive −130°C. for a "few hours," −24°C. for 12 days, and −10.6°C. for 24 days.

In gastric juice the bacilli die in 15 to 20 minutes but the spores are not destroyed.

In fairly thick blood or spleen smears dried on glass slides and kept in a room, a few bacilli of the many thousands present may still be alive at the end of 18 to 20 days (Frothingham), and Momont says that in dried rabbit's blood at room temperature, they will remain alive for 57 days. It is well known to all bacteriologists that spores dried on silk thread and kept in the laboratory without special precaution against diffuse daylight, retain their vitality and virulence for many years (20 at least) and perhaps indefinitely.

Sunlight at 25° to 30°C. kills bacilli in dried blood in 6½ to 15 hours according to the thickness of the layer, while spores dried on silk threads will withstand direct sunlight for 100 hours (Momont). According to Neumark, bacilli and spores on agar plates are killed in 20 to 30 minutes by sunlight, and diffuse daylight destroys bacilli in about 11 hours and spores in 2 days.

These and the following widely differing results of various experimenters are partly explained by the dissimilarity of conditions under which the observations were made. For example, it has been shown that different "races" or "strains" of anthrax bacilli have different resisting powers. Resistance also depends upon: the age of the spore (24-hour spores are said to be especially resistant); the temperature at which the spore was produced; the object upon which it was dried for experimentation, for in porous substances (silk, filter paper, cotton) organisms are less easily destroyed than on smooth surfaces (glass, stone). Spores on threads are said by one observer to be more resistant than spores in emulsion and another writer finds the opposite. It is said that 1 to 1000 *corrosive sublimate* will destroy anthrax spores in 20 minutes, but if albumen is present as in the blood, its action is untrustworthy. Fraenkel says spores are destroyed by a 0.5 per cent. solution of corrosive in 40 minutes and Geppert that they are not entirely destroyed by this solution in 2 to 3 hours, but that a 1 per cent. solution will kill them in 6 to 12 minutes, while Heider states that a 1 per cent. sublimate does not destroy in 3 hours.

In 5 per cent. *carbolic acid*, Koch says that the spores are dead in 2 days; Guttmann in 37 days, and Fraenkel that they are still alive after 40 days. Von Esmark found the spores of one strain were killed in 4 days, in another they were still capable of cultivation after 40 days' exposure to 5 per cent. carbolic; while if warmed to 37°C. they were destroyed in 2 to 3 hours and at 9° to 10°C. there were still living at the end of 10 days.

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Lysol (5 per cent.) kills the spores in 7 hours (Foth); *creolin* (10 per cent.) will kill bacilli in 10 to 20 minutes but spores are not harmed even by a 60 per cent. solution (Sirena and Alessi); *chlorine water* (Chlorwasser) (0.2 per cent.) destroys spores in 15 seconds (Geppert); *antiformin* (5 to 10 per cent.) completely dissolves bacilli and spores in a short time (Kolle and Wassermann); *formalin* (1 per cent.) kills spores after 2 hours, 2 to 5 per cent. in 1 hour, 10 to 20 per cent. in 10 minutes (Hammer and Feitler), undilulted formalin (40 per cent. formaldehyde) in 1 to 5 minutes; in a dilution of 1 to 20,000, formalin arrests the development of anthrax bacilli in bouillon cultures (Aronson).

In *soil* the bacilli die out fairly rapidly but the spores are said to survive for 15 years (Sirena, by Besson) and in water for 17 months. On the inside of a manure pile a temperature from 76.5° to 72°C. is said to destroy spores in 4 days (Pheiler, by Hutyra and Marek).

"Pickling destroys the bacilli in meat after 1½ months (Peuch), but it does not affect the spores (Abel). The drying and salting of hides does not destroy the attached spores which may remain virulent for 125 days on hides that have been treated with fresh milk of lime (Griglio). In sunlight, hides containing the bacilli are sterilized in 6½ to 7 hours while those containing spores are not sterilized even after exposure for 13 hours (Esmarch). Quick lime solution and lime kill the spores in the tanning process in 12 to 17 days. Infusion of quebracho bark* does not affect them in 12 days (Keszler). . . . as a matter of fact at the present time there is no method known for an effective disinfection of hides without injuring them for their technical utilization (Xylander) . . . . . Horsehair is effectively disinfected by dry heat at 110°C. followed by steam at 100C°. (de Rossi). The officially required disinfection of railroad stock cars in Germany with 5 per cent. "Kresulfol" (cresol-sulfonic acid) was found by Schnürer to be absolutely ineffective against anthrax spores and he recommended instead the disinfection with 0.5 per cent. formaldehyde solution" (Hutyra and Marek).

Anthrax in Animals.—The disease in animals is world wide in its distribution and has been known for centuries. The animals most susceptible to the natural disease are cattle, sheep, horses, hogs, and carnivora, in the order named, though infection in the last is exceptional, resulting as a rule from the ingestion of flesh of anthrax animals. Algerian sheep, domestic fowls (probably most birds) and the cold-blooded animals are not susceptible to the natural disease and are exceedingly resistant to artificial infection. Of the laboratory animals, mice (white and gray), guinea-pigs, and rabbits are readily infected even with minute doses. Rats are less susceptible, especially black rats.

Cattle in spite of their susceptibility to spontaneous anthrax, are frequently quite resistant to inoculation even surviving relatively large doses of virulent organisms. *Sheep* on the contrary seem to be as susceptible to

* Used for tanning purposes.

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inoculation as to natural infection. *Hogs*, although they may have the septicemic form, are much less susceptible and possess a relatively high natural resistance to inoculation (subcutaneous, intraperitoneal, spore feeding). In the majority of instances, the infection remains localized in the pharyngeal region.

Natural Infection in Animals.—In the herbivorous animals this usually takes place through food or water containing anthrax spores, and it is stated that large numbers of spores are usually necessary to produce infection in this manner. The food becomes contaminated by being grown on anthrax infected soil. The soil becomes infected in a variety of ways and perhaps the commonest way is from anthrax carcasses. If an animal dies at pasture or is dragged over the ground for burial, blood, urine, and feces, containing bacilli, escape from the natural openings and are scattered over the soil; but a more prolific source is the unburied or superficially buried carcass. Bacilli from an unburied carcass are soon distributed about by wild animals, dogs, birds, and insects and even fairly deep burial does not preclude upper soil contamination for bacilli and spores may be brought to the surface by rising ground water and perhaps by worms. The bacilli not being very resistant may be destroyed by sunlight or other adverse conditions, but under favorable circumstances, they develop into the exceedingly resistant spores which may remain virulent for years. Moreover under proper conditions, these spores may germinate resulting in innumerable bacilli which in turn sporulate. Spores may even reach the soil directly, for the intestinal contents often contains spores as well as bacilli; also from opened carcasses on the way to interment, for in the opened carcasses spores are frequently formed shortly after exposure to the air. Winds may carry dry spores to neighboring localities and rains wash them into brooks and streams. Thus arise "anthrax pastures" and "anthrax fields," and animals grazing over them may become infected; also crops grown on them may become contaminated with spores and if sent to other localities may be the cause of new centers of infection.

Anthrax infection in animals is recorded from fields contaminated with dust blown from a Persian wool factory and a horsehair factory (Oliver); Soil, food, and water also become contaminated by the "drainage water and waste from tanneries, wool-works and horsehair mills" (Hutyra and Marek). by fertilizers made from animal tissues; by the feet, bill and vomitus of buzzards; and the feet of flies and ants which have fed on anthrax carcasses. The material regurgitated by such flies and their feces (also the feces of the house-fly fed with spores) contain anthrax organisms; the feces of buzzards do not (Dalrymple, Mitzmain, Darling and Bates, Graham-Smith). Dalrymple made feeding experiments with the turkey buzzard (Cathartes auro), the carrion crow (Catharista atrata), the dog, pig, cat, oppossum (Didelphys virginiana) and the common fowl. He found *no* anthrax infection in the *feces* of buzzards and crows, but little in the *stomach* (24 hours after feeding

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spores) and none further on in the intestine. On the beak and feet virulent anthrax bacilli were found for 48 hours after feeding anthrax rabbits, and in the vomitus of buzzards 2 hours after such feeding. Dogs fed with spores showed virulent anthrax in their feces for 6 days; pigs for 5 days; cats and the oppossum 3 days and the common fowl 48 hours.

Food in stables may become infected by drying anthrax hides in hay lofts and other places where the blood from the hides may drop onto hay or other food; also the blood or discharges of anthrax animals may contaminate food. Bone meal made from anthrax carcasses and prepared foods may also contain infection. Stockman notes a great decrease in anthrax in England from April to September when animals are apt to be at pasture and a sudden increase after the time when they are returning to artificial food. He therefore attributes outbreaks less to pastures than to food stuffs (cake) and has known them to follow distribution of Soya beans. In one instance, Dr. Peters and I thought infection resulted from refuse from a carpet factory used for bedding.

Infection may also occur from the bites of flies which have previously sucked the blood from an anthrax animal, thus giving rise to the carbuncular form (Dalrymple). Mitzmain showed experimentally that anthrax could be transmitted by Stomoxys calcitrans and Tabanus striatus, though not with regularity. He found that the feces of these flies contained virulent anthrax bacilli for 9 days after sucking the blood from an anthrax animal. Cultures obtained later than 9 days after feeding were avirulent, though "all of the oridnary cultural and morphological tests were positive from the ninth to the twentieth day with material obtained from Stomoxys calcitrans and from the sixth to the tenth with material obtained from Tabanus striatus." The experiments were carried on for 20 days. Spores were not demonstrated either in the body of the flies or *fresh feces*. The possibility of spores developing in older fecal deposits was not included in these experiments.

Symptoms in Animals.—The incubation period after ingestion of spores is from 3 to 10 days. The spores are not destroyed by the gastric juice and pass into the intestine where they develop into bacilli, multiply and invade the blood by way of the lymph channels. Thus the disease becomes a true septicemia and the animals may die in a short time without showing very characteristic symptoms.

The *peracute* form simulates cerebral apoplexy and is seen most frequently in sheep. The animal suddenly falls in convulsions, blood oozes from the nose, mouth, and anus, and death occurs perhaps within an hour.

The *acute* and *subacute* forms are much more common. Symptoms are frequently not observed; the animal appearing well in the evening is found dying or dead in the morning, or the disease may last for 2 or 3 days, and in exceptional cases even longer. If symptoms are noted, they are usually those of an acute infectious disease not especially indicating its specific

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character. In some cases, more especially in the *horse*, rapidly growing œdematous swellings, hot, doughy or dense, may appear in any region of the body but are more apt to be on the neck, breast, flank, or lumbar region. When on the neck there is usually a more or less severe œdema of the glottis. In horses, also, there is usually a severe spasmodic colic.

Milk secretion is suspended early in the disease or, if secreted in small quantities, is apt to be bloody. MacFadyean found many anthrax bacilli in the milk, drawn after death, of three cows. Moore found bacilli in the milk of a cow at the time of death, but was unable to do so "in the milk of cows in advanced stages of the disease."

Carbuncular anthrax is not so frequently seen in animals as in man, though Dalrymple reports outbreaks of this form in the lower Mississippi Valley during the period when the horse fly (Tabanus striatus) is most abundant. In his experience, while initial cases of intestinal anthrax frequently occur in the late spring or early summer, the disease is more prevalent later as the carbuncular form and is widely spread by the bites of various species of the horse fly (Tabanids) which have previously sucked the blood of infected animals, and the more plentiful the flies the more prevalent the disease.

Anatomical Changes.—The most conspicuous pathological conditions are a fluid, tarry blood, and a very much enlarged spleen, its pulp being dark and tar-like and usually very soft, even fluid. There may also be more or less extensive hemorrhages and œdematous gelatinous infiltrations in the subcutaneous and intermuscular connective tissue, the mesentery, pleura, pharynx, and epiglottis. The small intestine—rarely the stomach or large intestine—may be swollen and congested with sometimes areas of necrosis. The lymph glands are usually much swollen, moist and hemorrhagic, especially the mesenteric and those near gelatinous infiltrations.

In the *horse* and *pig* the chief lesions are frequently confined to the lymph glands and to gelatinous, hemorrhagic swellings around the throat. The enlarged spleen may be absent, especially in swine. The blood often contains *no* anthrax bacilli after death, but they are found in the lymph glands and œdematous fluid. In the *pig* there may also be necrotic areas—the size of a quarter—on the mucous membrane of the pharynx, with almost black centers; these are of much diagnostic value and there is no difficulty in finding bacilli in such foci.

Much has been written recently in Germany regarding *local anthrax* in *hogs*, especially lymph-gland anthrax, which does not terminate fatally. Animals, apparently perfectly healthy, are found in the slaughter houses showing nothing but a few somewhat enlarged, yellow to deep red lymphatic glands. This condition may be confined to a single node of a group or even to a relatively small portion of the node, but from these lesions—frequently with difficulty—anthrax bacilli have been recovered, although they could not be found in other organs or the blood, and moreover the Ascoli reaction failed

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except with material from these infected glands. Such infection is supposed to be caused by certain foods, especially fish-meal (Fischmehl).

Diagnosis.—The capsule which surrounds the bacilli in the blood is of great diagnostic importance, for it is believed that this is the only large bacillus found in the blood and organs of dead animals that has this peculiarity. The numerous large putrefactive or cadaver bacilli appearing in the blood and tissues of animals (cattle, horses, sheep and swine) shortly after death, and even while moribund, have frequently been mistaken for anthrax. These organisms, however, do not possess a capsule.* It is therefore extremely important to stain such suspected material by a method which will bring out this capsule if present, for if special methods are not employed the capsules will not be seen. Numerous methods have been recommended, of which the first two following have proved very useful in my experience.

Smears of blood or tissue pulp, made fairly thick, should be air dried and fixed by passing rapidly three times through the flame, care being taken not to overheat or the capsules will be injured.

Johne's Method.—1. Stain the smear for about 30 seconds, heating gently, with 2 per cent. Gentian violet (dye 2 parts, water 85 parts, boil for 5 to 10 minutes and after cooling add 15 parts of 90 per cent. alcohol, mix and filter twice). 2. Wash in water. 3. Decolorize in 1 to 2 per cent. acetic acid 5 to 20 seconds according to the thickness of the smear. 4. Wash in water. 5. Mount and examine in water; if not sufficiently decolorized, remove cover slip with knife blade and repeat No. 3. The capsules are colorless and the bacilli violet.

The method I most frequently employ is simply a modification of the above; that is, decolorize with 95 per cent. alcohol instead of 1 to 2 per cent. acetic acid. Sometimes it is helpful to substitute for Gentian violet Loeffler's alkaline methylene blue, for with this stain the capsules, although they may be colorless by daylight, often show a delicate pink color if examined by artificial light. Fairly thick smears of heart's blood and liver pulp of an anthrax mouse made on a slide and kept for 5 years on the laboratory shelf (behind glass doors) still showed excellent capsules on most of the bacilli (Frothingham).

In view of these facts regarding the capsule and also the fact, above referred to, that cultures may be obtained from such dried material for about 20 days, I have found it convenient and practical for diagnostic purposes to recommend that several smears of blood or other material from suspected animals or man should be air dried (not with artificial heat or sunlight) on clean glass and forwarded to the laboratory. As it is unwise to open an anthrax-suspected animal, it is best to take blood from a superficial vein. The smear of blood should not be so thick that it cracks away upon drying,

* According to Noetzl, Berndts, Bongert, and others, some of these bacilli may occasionally show a capsule, though this is probably less pronounced than the anthrax capsule. It must also not be forgotten that in true anthrax the capsule cannot always be demonstrated, especially in horses and carnivora.

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but it must be dried, for if at all moist, other organisms which may be present will multiply rapidly and obscure cultures and microscopic preparations. At the laboratory, a portion of the blood is softened with sterile water and smeared in various quantities over several slant agars, the object being to obtain isolated colonies. Any suspicious colonies or growth appearing after 12 to 24 hours' incubation (see page 159) may be further tested in mice and subcultures. Another portion of the softened blood is smeared on cover glasses and stained for capsules, or one of the original smears is stained.

MacFaydean recommends staining air-dried and gently fixed smears with a 1 per cent. aqueous solution of methylene blue for a few seconds and describes a peculiar color reaction of the amorphous material in the smear, not necessarily in the immediate vicinity of the bacilli but probably derived from their capsules. This in smaller or larger granules or masses takes on a "violet" or "reddish purple" color which is in sharp contrast to the blue bacilli and nuclei and is especially well seen by artificial light. He considers the reaction of much diagnostic value as it occurs in smears of blood, œdema, and organ pulp of anthrax animals and not in similar preparations from animals dead of other diseases.

Frequently pure cultures of anthrax may be obtained from much contaminated material by heating a 24-hour mixed culture to 80°C. for ½ hour and plating or streaking out on agar.

Another good practice is to forward the ear of the suspected animal to the laboratory where blood may be pressed from the vessels for microscopic examination and cultures.

Ascoli Reaction.—Ascoli has worked out a precipitine reaction which has met with much approval and seems especially useful since it can be employed with small portions of organs and in cases where putrefaction is so advanced that bacteriological verification is impossible. It is said that this reaction is not effected by preserving anthrax tissues in glycerine, alcohol, or even formalin, and that the method may be used to detect anthrax hides and food stuffs (sausage meat, etc.).

The complement fixation method has been recommended, but does not seem to be as useful as the Ascoli test.

Prevention.—Grazing should not be permitted on lands known or suspected to be sources of infection, though it is frequently possible to fence off certain stagnant pools or other suspicious zones. Usually the regions which harbor anthrax infection are swampy or peaty, subject to overflows, the soil loose and warm and resting perhaps on impervious substrata, also naturally wet regions (fens, deltas, bottom lands) which, however, make available pastures in dry seasons. The only rational treatment of such lands is thorough drainage and proper cultivation, and they should not be used again for grazing purposes for several years. In this connection it is important to remember that part of the life history of various tabanids is semi-aquatic,

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and drainage therefore greatly reduces the number of these insects and hence tends to eliminate one source of the spread of anthrax.

If an animal dies at pasture great care must be taken to avoid contamination of the ground with its blood or discharges, and other animals should be immediately removed. The carcass should not be skinned or opened; if possible it should be burned, otherwise covered with powdered unslaked lime and buried at least 6 ft. deep in an unfrequented, fenced-off place, and ground known or suspected to have been contaminated with discharges should be burned over. If a carcass has been attacked by dogs, wild animals, buzzards, crows, etc., the danger of spreading infection is much increased; such creatures should be killed when possible and considerable ground in the vicinity of the carcasss burned over.

Suspicious food supply must be discontinued and destroyed. If an animal dies in the stable, everything suspected of being contaminated with its discharges should be thoroughly disinfected, burned if possible.

Preventive Inoculation (*Pasteur*).—This is very successful, is widely used and consists in the inoculation of two attenuated cultures. The first (Vaccine I) is very much attenuated and is only virulent for white mice, not uniformly for guinea-pigs. Vaccine II, given 12 to 14 days later, is virulent for guinea-pigs but not with regularity for rabbits. Active immunity is established 12 to 15 days after the second inoculation and lasts for 10 to 12 months. It may be transmitted to the fetus but is of relatively low grade and of short duration. The mortality from anthrax in cattle is about 0.5 per cent. of the treated animals (somewhat higher in sheep), and the death rate from anthrax on infected farms is reduced to 1 per cent. or less (Stockman).

In regions where infection is known to exist protective inoculation should be carried out early each spring so that immunity is established before the animals are turned out (Dalrymple).

An *anti-anthrax serum* (Sclavo) is obtained from artificially immunized animals which gives *passive immunity* for about 10 days (rarely 2 to 3 months), long enough, however, to protect against exceptional risks of infection. For example, if an animal dies among others and its blood or other infectious material split, the exposed animals may be given the serum which should protect them while disinfection is being carried out or an active immunity is being established. It is also said that as a cure in animals Sclavo's serum has frequently been used with success even after the bacilli have appeared in the blood.

Simultaneous preventive vaccination has been successfully employed since 1902 in "hundreds of thousands" of animals (cattle, sheep, horses, swine) in many countries—Argentina, Uruguay, Germany, Austria-Hungary, Roumania, etc. It is a combination of *active* and *passive* immunization produced by the simultaneous injection of serum and an attenuated culture of about the virulence of the Pasteur Vaccine II. The advantages over the Pasteur method are: only one treatment required; a more rapidly acquired

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immunity (10 to 12 days after inoculation) which is of a high grade and lasts for 1 year and perhaps longer.

General preventive measures[1] are of much importance. Animals from infected districts should be quarantined and the importation of hides carefully controlled and prohibited from infected regions. Imported hides, calf skins, hide cuttings and parings, horns, hoofs, hair, wool, bristles, glue stock, etc., should be disinfected if possible and certainly the waste from factories handling such products sterilized before it is permitted to reach fields. As before pointed out, there is at present no known method of sterilizing hides without injuring them, but the Bureau of Animal Industry in Washington is working on this problem and it is hoped that a method will soon be devised.*

Anthrax in Man.—The intestinal form of anthrax is rare in man and is caused by the ingestion of infected foods, such as sausages which have been made from the meat of an anthrax animal, or the flesh of such an animal insufficiently cooked; it may also arise from eating with hands not properly cleansed after handling infectious material. Pulmonary anthrax is more common and arises from the inspiration of dust from infected wool, rags, hair, etc., which contains anthrax spores—hence the names "wool-sorter's and rag-picker's disease." The mortality from pulmonary anthrax is about 50 per cent., and less than this in the intestinal form. The more usual form in man is the carbuncular, the so-called malignant pustule, and is the result of skin infection, bacilli or spores gaining access through cuts or abrasions of any kind, and probably in some instances directly introduced by insect bites. The pure septicemic form is rare. It is, therefore, evident that certain occupations favor infection and the disease is usually met with among workers on hides, hair, bristles, wool, etc., and in laboratory workers, veterinarians, meat inspectors, herders, farmers, cattle men, and butchers.

Oliver reports that in certain woolen districts in England where much foreign wool was handled anthrax was quite common, while in other parts of England and Scotland where only colonial or home-grown wool was manipulated the disease was " conspicuously absent." Legge reports that there were 261 cases of anthrax in the factories and work shops of England in 6 years (1899–1904); of these, 88 occurred in worsted and wool factories, 70 in horsehair and bristle works, 86 among workers on hides and skins, and 17 in other industries 67 cases were fatal. "The disease has appeared mostly in wool-sorting, wool-combing and spinning industries, in the manipulation of horsehair for stuffing chairs and mattresses and in the preparation of

* Since this was written the U. S. Department of Agriculture has published the results of experiments by F. W. Tilley: "A Bacteriological Study of Methods for the Disinfection of Hides Infected with Anthrax Spores" (Journal of Agricultural Research, Vol. IV, No. 1, April, 1915). The conclusions are that although "the Schattenfroh method cannot be regarded as perfect, it nevertheless seems to be far superior to other methods and well worth a trial as a standard method for the disinfection of hides."

The Schattenfroh method consists of a 48-hour exposure of the hides to 2 per cent. hydrochloric acid and 10 per cent. sodium chloride. Experiments are still going on to ascertain the effect of this disinfection upon hides as regards tanning, but it seems at present that the method has no injurious effect upon hides or leather.

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bristles for brush-making" (Oliver). Only a very small percentage of the persons employed became infected.

Hope and Hanna report 60 cases of anthrax in Liverpool which occurred during the 8 years previous to 1912. Of these, 36 individuals unloaded or carted hides at wharves, 13 handled wool and 6 horsehair. In 1910 there were only 9 cases reported in England and but 11 in 1911 (Thompson).

In the United States the disease is reportable in only about half of the states and general statistics are lacking. There is no doubt, however, that a certain number of cases occur every year. Thus in Massachusetts for a number of years there has been an average of two deaths annually. In the registration area of the United States there were 18 deaths in 1912, and an annual average of 21 deaths from 1906 to 1910. Ravenel reported 12 cases in Pennsylvania in 1892 among tanners of hides from China; Thompson, three cases in the woolen carpet industry, and three cases were reported in New York state in the year 1911–1912.

A record of the reported fatal cases of anthrax is kept by the United States Bureau of the Census. In the 4 years 1910 to 1914, 79 such fatal cases were reported. As in other countries, Andrews points out that most of these cases occurred among persons whose occupations brought them in contact with infected material. The cases included farmers, tanners, leather workers, brush makers, workers on hair and haircloth, and assorters of hides. The most frequent source of infection, Andrews reports, was among assorters of hides. Other sources of infection assigned were: fine wool or other animal hair, diseased cows, rags, old clothes, infected knives, and mosquito bite.

In Germany, where records are more carefully kept, there were in the period 1900–1908, 1042 cases of anthrax in man and 49,458 in animals. In 1910 there were 287 cases of which 40 were fatal; in 1911, 281 cases with 40 deaths, and in 1912, 274 cases with 36 deaths. In England, in 1910, 51 cases and 9 deaths were reported; in 1911, 64 cases and 11 deaths, and in 1912, 47 cases and 6 deaths. In France, in 1910, 1911 and 1912, there were 54, 42 and 38 cases respectively reported. John B. Andrews states that not all these cases were due to the worker's occupation. He reports that in Germany out of 287 cases that occurred in 1910, 24 were among persons whose occupations were not considered a factor in the disease, and points out that many cases occurred among persons of agricultural occupations. In Italy, 1890–1900, there was an annual average of about 2100 cases among people, and in Russia, 1904–1909, an average of 16,000 per year (Kolle and Wassermann).

Anthrax in man is observed in the following forms: *External Anthrax:* (*a*) Malignant Pustule, (*b*) Malignant Anthrax Œdema. 2. *Internal Anthrax:* (*a*) Intestinal, (*b*) Pulmonary (Wool-sorter's Disease).

Symptoms. 1. *External Anthrax.* (*a*) *Malignant Pustule.*—The incubation period is from 1 to 3 days and the point of infection is usually the

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hand, arm, face, neck, etc., *i.e.*, exposed parts likely to come into contact with hides and parts frequently scratched with the finger nails. Often within a few hours after infection there is itching and uneasiness and the gradual formation of a small hard pustule which soon becomes vesicular and discharges a bloody serum, and within 36 hours there is a dark brownish eschar. Inflammatory induration extends around the point of infection and at a little distance there may be a series of small vesicles. Œdema causes marked swelling of the parts, the inflammation extends along the lymphatics and the neighboring glands are swollen and sensitive. The temperature rises rapidly at first, later falling and frequently becoming subnormal. There is much variation in the severity of the cases; death may take place in 3 to 5 days; where recovery occurs the constitutional symptoms are less marked, the eschar gradually sloughs and the wound heals. In the mildest form there may be but little swelling, the inoculation papule rapidly vesiculates and the resulting scab separates in a few days (Osler).

(*b*) *Malignant anthrax œdema* is observed in the loose connective tissue of the eyelid, hand, neck, thigh, etc., and is characterized by absence of redness, papules and vesicles, but by the presence of extensive œdema—following rather than preceding constitutional symptoms—which is not well circumscribed, spreads rapidly and is apt to terminate in sloughing and gangrene. The mortality in infections about the head is 26 per cent. (Narsarow) and infections of the legs 5 per cent. (Osler, Thompson).

2. *Internal Anthrax.* (*a*) *Intestinal.*—This form results from the ingestion of anthrax-infected foods (see page 168), though it may follow the external type if the bacilli are carried to the mouth from the external lesions. The symptoms are those of intense poisoning: chill, followed by fever, vomiting, diarrhea or constipation, pain in various parts of the body, dyspnœa, and cyanosis. There may be hemorrhages from the mucous membranes; the spleen is enlarged and the blood is dark and does not coagulate after death. Butler and Huber mention an instance where 25 people became infected after eating meat from an anthrax animal and six died in from 48 hours to 7 days (Osler).

(*b*) *Pulmonary Anthrax, Wool-sorter's Disease.*—This results from inhaling dust containing anthrax spores. The symptoms correspond more or less to those of the intestinal type with the addition of rapid respiration, cough and pain in the chest. Death may occur in 24 hours; when the disease is of longer duration there may be delirium and unconsciousness (Osler). Oliver notes that although this form of anthrax is usually fatal in 2 to 4 days, there is an absence of the severe symptoms usually accompanying acute infections, and "the characteristic feature is the rapidity with which collapse sets in."

Diagnosis.—The symptoms and history are sufficient to lead to a suspicion of anthrax infection and laboratory methods must be resorted to for diagnosis (page 165). Anthrax bacilli may be demonstrated in the fluid from the

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pustules, the œdema, sputum (in the pulmonary form), and, during the last stages of the disease, in the blood.

Treatment.—The use of actual cautery on the malignant pustule has been recommended, also the removal of the whole infected area and neighboring lymphatics with the knife. Local development of the bacilli is said to be retarded by subcutaneous injections of 3 per cent. carbolic acid or 1:1000 corrosive sublimate repeated two or three times a day, at various points in the neighborhood of the pustule. But these methods cannot be regarded as successful.

On the other hand, there is much evidence that no surgical intervention whatever gives the best results, the treatment simply consisting of maintaining the general health and protecting the local lesion by aseptic or antiseptic dressings. For example, Leland* has tabulated the cases occurring at the Massachusetts General Hospital, Boston, during the past 36 years. These records show 23 cases: 7 of these were operated upon and 3 died (42.8 per cent.); the other 16 were not operated and all recovered. Others reporting successful non-operative treatment are: Muskett 50 cases, Müller 13 cases, Remstedt 7, and Savini 5, all recovering, Creite 13 recoveries and 1 death, and Graef 384 cases with 20 deaths.

Treatment with Sclavo's serum (page 167) is successfully reported both in local and general infections. Thus Italian statistics show a mortality of 6.09 per cent. of the treated against 24.16 per cent. for the rest of Italy; and in England, including cases already moribund, 7.4 per cent. mortality among the serum-treated, while in previous years (1889–1903) without serum it was 26.5 per cent. In some cases this treatment has proved successful even after anthrax bacilli have invaded the blood.

Prevention.—It is evident from the foregoing discussion that anthrax would not occur in certain trades if all dangerous material were properly disinfected before manipulation by the workers. To accomplish this without injuring the product for its intended manufacturing purpose is the difficulty not yet overcome in many lines of industry. Until adequate methods of disinfection are perfected and enforced other means of prevention must be recommended.

Dangerous dust should be drawn from the sorting tables, etc., by exhaust ventilation downward and so away from the face of the worker. The workers themselves should take every precaution against infection according to their special employment: proper clothing, dust masks, strict personal cleanliness, hands thoroughly washed before handling food, meals not eaten in dangerous surroundings, cuts and abrasions carefully protected, hides handled with gloves and not carried against bare neck, shoulders or arms.

Preventive inoculation may also be seriously considered for workers in the dangerous trades, though as yet statistics are not available along these lines.

* Geo. A. Leland, Jr., "The Treatment of External Anthrax." To be published.

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*Legislation.**—"In order to minimize the danger to workmen, sanitary measures are prescribed by law in a number of European countries, for instance in England, Germany, France, Belgium and Holland. Fairly effective legislation on anthrax is to be found in England where for a number of years the government factory inspectors have had the valuable coöperation of the Anthrax Investigation Board. In Germany the crusade against anthrax is of more recent origin; statistics are being kept there since 1910. Each case is thoroughly investigated and the source of the disease is traced as far as possible. In both countries notices are posted in factories calling attention to dangers and precautionary measures.

In England the law of 1901 referring to work on imported hides and skins makes provisions for keeping the workers' food and clothing in a clean place. Water, soap towels and nail brushes must be furnished, also overalls and gloves if dangerous hides are handled. Every workman having an open cut is required to report immediately to the foreman and to leave the shop until the cut is healed. Still more careful regulations are prescribed for work on hair imported from Russia, also from China, Siberia and other Asiatic countries. Non-disinfected material must be kept in a separate room to which no food or drink may be taken. Careful rules are prescribed for opening, disinfecting and handling materials for the removal of dust and for personal cleanliness. The precautionary rules of Germany and France follow very closely those of England."

* Since this chapter was written the editors received from John B. Andrews the above summary of legislation on anthrax and also some of the statistics on anthrax in the United States, England and Germany cited on page 169.

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SECTION II

PARASITES AND OCCUPATION

BY BAILEY K. ASHFORD, M. D., Porto Rico

Whatever may be our idea of what should constitute "occupational diseases," it is undoubtedly true that we have only recently begun to protect that most humble and yet most important of all laborers, the tiller of the soil. The diseases to which he is by reason of his occupation especially exposed, malaria, uncinariasis, schistosomiasis, amebic dysentery, filariasis and ascariasis, are briefly summarized below. As our National tendency is toward the specializing of labor, toward the refinement of the raw product, toward the factory, so the southern countries are tending toward the refinement of agriculture, toward the production of the largest output and highest type of vegetable life where soil and climate best favor these products. But just as it was impossible to build a Panama Canal until the parasitic foes of man were conquered, so will it be impossible to reap the fullest harvest from tropical and subtropical lands, to colonize in its broadest sense, until we learn to protect ourselves from the luxuriant parasitic life which is favored, as are valuable crops, by a propitious climate. With apologies to those who consider a tropical climate per se an obstacle to the well-being of the white man, it is suggested that judgment be deferred until the effect of recent sanitary reforms has impressed itself upon those who, living in warm climates, seek to avoid the diseases heretofore considered unavoidable. Indeed many so-called "tropical diseases" might better be termed "rural diseases," so extensive also is their domain in certain country districts of the temperate zones. Already from tropical and subtropical lands has sprung the efficient prophylaxis of malaria, responsible for the agricultural depression of many sections of our south, of yellow fever, and other diseases which wave-like have beaten upon our shores, and have even, as in the case of "hookworm disease," successfully invaded nearly the half of our country in so subtle a form as to have been unrecognized for probably a hundred years. Thus we may consider the tropics as the original home of most diseases due to animal parasites. There still they best flourish and yield their greatest percentage of inefficiency for labor, due first to heat and humidity and second to ignorance of modern individual hygiene, upon which all true State and National hygiene is most firmly based. The essence of sanitation is education in preventing communicable disease. In emergencies, imposed by force of law, the natural evolution of sanitary living

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must ordinarily spring from the intelligent coöperation of the good citizen whose will in the form of a comprehensive sanitary organization should control the ever-present ignorant, careless or criminal spreader of disease and death. In a perusal of these pages it will be seen that by a careful observance of the very simple prophylaxis of two notoriously dangerous enemies of agricultural countries, malaria and uncinariasis, we go far toward exterminating on the one hand dengue, filariasis and yellow fever, and on the other, typhoid fever and the dysenteries, not to mention other communicable diseases less directly affected.

PROTOZOA

Malaria.—An infectious disease caused by the introduction into the human blood through the bite of certain anopheles mosquitoes, and by them alone, of the sporozoites of one of three species of hemosporidia—Plasmodium malariæ, Plasmodium vivax and Laverania malariæ. The ameboid parasite enters the erythrocyte, transforming its hemoglobin into a black pigment "melanin," and finally, reaching its maximum growth, sporulates, liberating its daughter cells, or "merozoites," to attack fresh red cells. In rupturing its containing cell it discharges a hemolysin on the one hand and a pyretogenous toxin on the other, the latter provoking the typical sequence of chill, fever and sweat with their clinical accompaniments. The predominant brood of P. malariæ is liberated every 72 hours, that of P. vivax in 48 hours, and that of Laverania malariæ in 24 to 48 hours, these three varieties constituting the quartan, tertian and æstivoautumnal forms of the disease, the latter being the tropical and most dangerous type. Typically there is an intermittent afebrile period between each brood, but in æstivoautumnal fevers this period, at best but short, may be merely indicated by a remittance if, indeed, a most irregular or continued fever be not seen. Craig believes in two varieties of æstivoautumnal parasites, one the subtertian the other the quotidian. As the antitoxin of the host develops, which may eventually bring about a cessation of symptoms, gametocytes or sexual forms appear which, ingested by a fresh mosquito of the proper species, undergo development therein and give rise to infective sporozoites in about 2 weeks. Sporulation is not ordinarily seen in the peripheral blood in infections by Laverania malariæ but in the internal organs, especially the spleen, liver and bone marrow. Here in these filter-like organs, as well as in the brain, intestines, pancreas, etc., the parasited erythrocytes damaged by the toxin tend to stick to the walls of capillaries likewise affected and cause emboli (Castellani and Chalmers) which are productive of serious and often fatal accidents. All forms are capable of producing "pernicious malarial fever," but especially L. malariæ, 1 per cent. of æstivo-autuminal infections forming this type. Relapses occur, alleged to be caused by the

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return to asexual activity of gametocytes dormant in the blood and internal organs, especially the spleen (latent malaria). Anything tending to weaken the acquired relative immunity may precipitate an exacerbation, such as excesses, exposure to hot sun, fatigue, etc. Children are the chief reservoirs of gametocytes, and from the examination of their blood and spleen Sir Ronald Ross constructs with other factors his "endemic index." This latter author has shown that when the number of anopheles capable of transmitting malaria is reduced below a certain ratio malaria will not develop. The necessary factors for endemic malaria are (1) certain anopheles in an active state, (2) infected persons, and (3) individual susceptibility. Malaria is not found usually above 62 degrees north in the Eastern and 45 degrees north in the Western Hemisphere. Men engaged in ditching in railway construction, soldiers, sailors in port and agricultural laborers in infected districts are very liable to contract malaria. This disease in all time has been the greatest obstacle to colonization of the tropics by the white race; it has transformed fertile, rich and populous regions into lonely wastes; it has rendered the development of valuable natural resources impossible and by decimating armies has defeated the highest hopes of nations in critical struggles. In the Canal Zone the French lost 22,000 with an average working force of 10,000 laborers. In the same length of time with an average force of 33,000 we have lost but 4000. The sanitation of the Zone cost but about $3.50 per capita of population per annum or about 1 per cent. of the total expense of building the canal. To-day the Zone is as healthy as many northern cities. In Havana the mortality from malaria fell from 350 a year to 10, and yellow fever disappeared. In Rio de Janeiro there has been a fall in mortality from malaria from 2235 in 1891 to 176 in 1911, and from 4456 deaths from yellow fever in 1891, that disease disappeared completely in 1908. All of this is the result of the organized application of modern prophylaxis.

Prophylaxis.—There are four essentials: (1) prevention of schizogony in man and the cure of all infected, (2) prevention of mosquito breeding, (3) protection of man from their bites, (4) education of the public. The first is accomplished by the use of quinine. The second may be divided into (*a*) removal of water in which mosquitoes breed, (*b*) rendering water unfit for breeding. Removal of water is best carried out by drainage, and tile drains are especially effective and the most economical in the long run. Where drainage or filling is impracticable water is best rendered unsuitable for breeding by covering it with oil. Larvicides of various kinds, Phinotas oil, etc., may also be used efficiently. One ounce of crude petroleum to 15 sq. ft. of water is recommended applied not less than once in 2 weeks, better once a week. To drainage must be added the removal of mosquito shelter (jungle growth, long grass, shrubbery, etc.) for ¼ mile from habitations. Mosquito brigades, or a corps of men whose business it is to prevent mosquito breeding in or near human dwellings by the prevention of water collection

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in little pools, in old cans, depressions, etc., is a valuable adjunct. Wire screens to windows and doors are of great value in excluding the mosquito, as are head nets, mosquito bars, etc. The mesh should not be less than 16 wires to the inch. The gametocyte population should be segregated and all sick of malaria isolated and screened. In the Canal Zone, General Gorgas depends on the destruction of the habitat of anopheles within 200 yds. of all dwellings, as well as of harbors for adults (shrubbery, long grass, etc.), screening, the use of crude oil in temporary pools and of Phinotas oil, or copper sulphate for grass and water containing algæ. The supply of prophylactic quinine to those who will voluntarily take it is an important method employed, but he depends on draining (especially on tile under-drains) for permanent results. He considers 1½ per cent. morbidity an indication of an improper working of his system. For prophylaxis Koch's method of 1-gram doses every eighth or ninth day and Celli's method of 0.4 daily are the most popular.

Treatment of Malaria.—Half a gram of quinine every 3 hours until parasites disappear is sufficient. It should be followed by 0.30 to 0.60 a day for a week thereafter, and subsequent to this 0.60 once a week for at least 2 months. It should never be given in pill or tablet form, and is best administered in solution with yerba santa or chocolate syrup. The severe forms may require hypodermic injections of quinine three times a day in ½-gram doses, better intravenous injections of even larger doses well diluted in salt solution. Avoid administering quinine in the height of an attack if possible. Substitutes for quinine are unreliable. Warburg's tincture is of little value and arsenic of less, save for subsequent anæmia.

Amebic Dysentery.—While frequently found in temperate zones, it is really an endemic of the tropics. It is due to a protozoon, Entamœba histolytica, usually from 25 to 40 microns in diameter, with a clear hyaline refractive ectoplasm and a finally granular endoplasm containing vacuoles, a variable nucleus (best studied by staining with Mallory's iron hematoxylin of wet fixed smears in Schaudinn's sublimate alcohol), and, almost constantly, red blood corpuscles. Under certain conditions it becomes resistant with the formation of a cyst containing four nuclei, a characteristic which is responsible for the term "E. tetragena." A precystic small form is often seen. The parasite enters the submucous tissue of colon, lower ileum and rectum, undermining the mucosa and producing large irregular ulcers usually placed at right angles to the bowel. It all too frequently passes by way of radicles of the portal vein to the liver where it sets up single or multiple abscesses. The majority of infections are liable to be undiagnosed, even in the presence of serious lesions, owing to latency and, frequently, colorless symptomatology. Accustomed to view "dysentery" as a disease whose syndrome is marked griping and tenesmus with frequent, small, muco-sanguinolent stools, the physician is often unprepared in the absence of the acute stage, for an alternate looseness and constipation of the bowels without marked constitutional

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involvement. The practitioner who lacks time or experience for the comparatively simple distinction between entamœba coli, a harmless commensal and E. histolytica, should refer the examination of the stool to a competent protozoologist. Recently, and this is what makes correct diagnosis so extremely important, we have been furnished by Leonard Rogers, with what bids fair to be accepted as a true specific for this dangerous and insidious disease, emetine hydrochloride. James has just recounted degenerative changes in the entamœbæ immediately following its use which account for the remarkable and prompt clinical results. Following the usual treatment, absolute rest, liquid diet, preliminary emptying of the bowel by salts or oil, and morphine, ½ grain of emetine hydrochloride is administered hypodermically three times a day for 2 or 4 days. It has been objected that the entamœbæ do not always disappear under this treatment, but the writer can testify to almost uniformly favorable clinical results. James counsels the use of 9 to 12 grains in as short a time as possible, compatible with individual tolerance, to forestall relapses. A daily dose of from 2 to 4½ grains can be reached in 3 days. This treatment should be followed by a heaping teaspoonful of bismuth subnitrate every 3 hours, which usually causes prompt disappearance of the parasite, and is supposed to deprive it of certain substances necessary for its nutrition by combining with sulphur in the intestinal canal. The prophylaxis consists in the avoidance of contaminated drinking water, raw vegetables and fruits. Sight should not be lost of the fact that certain authorities believe that it is in part if not wholly a contact infection.

NEMATODES

Uncinariasis.—This is an infectious disease caused by the presence in the small intestine of man of a sufficient number of adult nematodes of the subfamily Uncinariinæ, species Anchylostoma duodenale and Necator Americanus, to overcome his relative racial or individual resistance. The worm, from ½ to ¾ in. long and of the thickness of a pin sinks its buccal armature into the intestinal mucosa causing solution of continuity for the admission of bacteria and at times local hemorrhage, feeds upon the epithelium and produces a hypothetic toxic substance probably responsible for many clinical features of the disease. The ova do not hatch in the body of the host but, passed with the feces upon a damp soil, at a favorable temperature evolve into ensheathed larvæ which infect a new host by penetration of the sound skin, usually of bare feet, causing "ground-itch." From this point they migrate to their site of election in the jejunum. The infective larvæ are usually found upon the soil in little nests, larger than, but corresponding to, the site polluted by ova-bearing feces. In endemic areas of the tropics and subtropics there is perennial infection of agricultural laborers. In temperate zones, in mines and tunnels, and in the summer throughout farming districts,

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FIG. 2.—A SAGITTAL SECTION OF NECATOR AMERICANUS ATTACHED TO THE INTESTINAL MUCOSA.
Microphotographs made by the late Dr. W. M. Gray, U. S. Medical School, of sections belonging to Major B. K. Ashford, member of the late Porto Rico Anæmia Commission.

FIG. 3.—STAGES OF DEVELOPMENT OF OVUM OF NECATOR AMERICANUS.

Microphotographs by, Dr. Wm. Gray, Army Medical School of sections belonging to Major B. K. Ashford, member of the late Porto Rico Anæmia Commission.

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intensity of infection is only measured by the amount of surface pollution by ova-containing feces. Infected soil seems to become relatively innocuous after 6 months freedom from pollution. Shaded, moist soil in warm weather and density of population in a barefooted people who directly pollute the surroundings of their homes and their place of work are the determining factors in producing severe endemics, given introduction

Fig. 4.—Photograph of a patient of the Institute of Tropical Medicine and Hygiene of Porto Rico suffering from uncinariasis intense grade; hemoglobin 14 per cent., erythrocytes 751,000, leucocytes 17,000, case of several years standing. Photograph furnished by Major Bailey K. Ashford.

of carriers. When larvæ nests are few and far between, and ova deposited by carriers are scarce, a low endemic index may be reached capable only of producing carriers and even these quite accidentally. Regions thus lightly infected may become dangerous foci if their density of population is considerably increased by an unsanitary laboring class. Shoes are not always an efficient protection, as they are often flooded by muddy water. Light infections produce few or no symptoms, at best debility and disin-

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clination for work, with a little dyspepsia and faint pallor. From such light cases we may proceed, as subsequent reinfections are superadded, to the very gravest forms of anemia, accompanied by an effacement of character and energy and retarded mental reaction quite peculiar to the disease. The best treatment is the weekly administration of thymol, based on an adult dosage of 4 grams, in capsules, 2 grams at 8 and 2 at 10 A.M. on an empty stomach, preceded the night before by a light supper and a purge and followed at noon by a purgative dose of sodium sulphate. After three such doses the patient may feel reasonably sure of a cure of his disease as he will have usually expelled over nine-tenths of his parasites, but persistent thymolizing may be required to rid him of all. Necator Americanus will not be ordinarily expelled by filix mas, the preferred anthelmintic in Europe. Betanaphthol in 2-gram doses may be administered as recommended for thymol.

Prophylaxis.—Uncinariasis is to-day one of the most serious obstacles to agricultural labor in the world's tropical and subtropical belt. Indeed, much of the "chronic malaria" of our South is uncinariasis. It has threatened the completion of tunnels and terminated mining operations; it has empoverished not only agricultural laborers but their employers; it has laid a benumbing hand on the growing country youth and, at times, it has been largely responsible for the poverty, ignorance and invalidism of a full four-fifths of the working classes of an entire country. The control of uncinariasis depends first on education in individual hygiene, second on the treatment of worm-sick, and third and least efficacious, on sanitary law. The first two are best served by the free rural dispensary. As the Porto Rico campaign was the first of its kind and has served as a basis, at least in part for subsequent ones in other countries, including our own, we will refer to its results. The hemoglobin percentage of 579 country people averaged 43.09 before treatment in 1904. In 1913 after the treatment of about one-half the island population of a million, the examination of 579 of the same type of people rendered an average of 72.22 per cent. hemoglobin. As the hemoglobin percentage is roughly equivalent to percentage of efficiency for labor, it can be truthfully said that treatment and education alone have raised the laboring efficiency of the working class by nearly 68 per cent. By far the greatest effect has been from treatment, for although shoes are more generally worn, privies are still scarce enough. The sanitary privy is the finality toward which all work should tend.

Filariasis.—The term usually signifies a condition accompanying the presence in the lymphatic system of man of a hair-like nematode worm, Filaria Bancrofti, whose embryos or microfilariæ appear in the blood. These cylindrical and ensheathed embryos, with blunt anterior and sharp posterior extremities, are 7 to 8 microns in diameter and 300 microns long, moving in graceful curves among the erythrocytes. Normally herding in the profoundest depths of the circulatory system by day, they invade the

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peripheral vessels by night, probably to better gain their intermediate host, certain species of culex and anopheles mosquitos. In the stomach of this insect their sheath is hemolyzed and, penetrating its wall, they develop in the muscles of its thorax, finally reaching labium and palps. When the mosquito thrusts its probosis into man, they escape by rupture of the distended tissue in which they are confined and enter the skin through the puncture as well as the unbroken surface in its vicinity. The parasite is generally distributed throughout certain districts of intertropical regions with a decided preference for hot lowlands. The typical lesion is a varix of lymphatics set in fatty tissue, and the seat of recurrent lymphangitis. Frequently localized in the vicinity of the external genitals the extreme may be reached by the formation of a huge varix involving the glands and tributary vessels of the abdominal and pelvic lymphatics. To Manson's theory of mechanical obstruction of lymph channels by adults, embryos and immature ova should be added a lymphangitis by the repeatedly awakened virulence of some strain of streptococcus, alone or in symbiosis with other bacteria among which may be mentioned Le Dantec's dermatococcus, or Dufougeré's lymphococcus. Clinically we recognize: (1) *Endemic lymphangitis* with red, tender lines in the course of certain lymphatics, inflammation of the receiving glands, severe pain and a sharp constitutional febrile reaction. Bubonic plague may at times be simulated and death occur. Of serious prognosis is intra-abdominal or intrathoracic abscess, an unusual complication. (2) *Lymphatic tumors*. Adenolymphocele, generally at the base of Scarpa's triangle, has the feel of a mass of soft rubber tubes and may be confused with hernia, lymphadenoma and lipoma. Pedunculated inguino-scrotal lymphangioma has only a superior pedicle and exactly simulates hernia; when inflamed, a strangulated hernia. (3) *Lymphatic varicocele* and *testicular lesions*. Acute filarial, often misnamed "malarial" orchitis, often causes, as do inflamed lymphatic varicocele and adenolymphocele, pseudorenal colic, a pitfall for the unwary tropical practitioner. Lymph scrotum, a cutaneous and subcutaneous infiltration is generally accompanied by hydrocele, often with thickened cartilaginous-like tunica vaginalis and a permanently damaged testicle and epididymis. (4) *Lymphorrhagias*. Chyluria is the emission of milky, greasy urine, and hematochyluria the passage of chylous urine mixed with blood. The pathologic basis for this phenomenon is the dilatation of the lumbo-sacral lymphatics with back flow into the lymph vessels of kidney, ureter and bladder. The attacks are usually irregular and embryos are apt to be found only in the first emissions of chylous urine. Lymphorrhagia externa is best seen on pricking a bulla of a lymph scrotum which allows the gradual filtration of quantities of lymph. (5) *Elephantiasis*. The skin is typically rough, tough, hard, covered with tubercles and often greasy. On section it is either like bacon rind, below which lies a yellowish gelatinous substance filling the connective-tissue spaces, or more fibrous, creaking under the knife. Such elephantoid tissue in marked cases hangs in huge folds and

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resembles elephant hide. This is the final result of a chronic filarial lymphangitis, often aggravated by acute attacks. The parts usually affected are the leg, foot and scrotum, but penis, labia majora, breasts, forearms and hands are not exempt. Some of these skin tumors are enormous, causing great deformity and even preventing locomotion.

Treatment.—There is no specific. Salvarsan, atoxyl and other arsenic preparations are useless. Filarial lymphangitis with its dependent end-products is best treated by removal to a northern climate. When acute, ice locally with complete rest in bed and appropriate symptomatic medication is all that can be recommended. Ichthyol locally seems to be of value. Extreme caution is necessary in dealing surgically with lymphatic tumors. They are most apt to become septic and are usually only a part of an extensive and hidden mass in the pelvis. Castration for filarial orchitis is usually unjustifiable. Elephantiasis is best treated by elevation, compression, a daily course of 30 to 90 hypodermic injections of 2 cc. of a 10 per cent. solution of fibrolysin and perhaps, if needed thereafter, the excision of skin strips. In the scrotum, vulva, etc., extensive removal and adjusting of healthy skin flaps is the best procedure. The prophylaxis is as for malaria.

Trichinosis.—Due to Trichinella spiralis, a nematode which passes its entire life cycle in man, rat and hog. The parasite is ubiquitous, but especially common among sausage makers. The larvæ encysted in the muscles, when the flesh is ingested, become mature worms whose embryos penetrate the intestinal wall and again reach the muscles. The chief reservoir of trichinella spiralis is the rat, which feeds on its kind and on scraps of infected hog flesh and on infected feces. Severe infections cause in man two stages of the disease; first a gastro-intestinal, and second a constitutional, with intense muscular pains, œdema, a sharp febrile reaction and eosinophilic leucocytosis.

Treatment.—Smart purging in the first stage or santonin and calomel. The treatment of the second stage is symptomatic.

Prophylaxis.—Meat inspection is impracticable as an efficient prophylactic measure. The larvæ die after cooking at a temperature over 160°F. or long-continued pickling. This is another argument besides plague for a rat warfare, especially in slaughter-houses, meat shops and the vicinity of hog pens. Hogs should not be fed on waste from salughter-houses, and should be kept away from privies.

Ascaris Lumbricoides.—A white or rose-colored cylindrical worm, 15 to 30 cm. long with a diameter of from 4 to 8 mm. The anterior extremity is blunt; the posterior pointed. The sexes are separate and the ova are ellipsoid, 50 to 75, by 40 to 50 microns, brown or amber-colored with two envelopes, the internal smooth and resistant, the external mammillated and easily detached. Habitat, small intestine of man. It is ubiquitous. There is no intermediary host, the ova developing after 15 days in water, but, being very resistant, they may reach maturity even after 5 years (Lecomte).

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While not mentioned in connection with occupations in the temperate zones, in the tropics it is exceedingly common among agricultural laborers from the use of fruits and raw vegetables. The recent investigation of the Institute of Tropical Medicine of Porto Rico, 1913, shows it to be the prevailing intestinal parasite of the coffee laborers, about 90 per cent. of 10,140 persons of all ages and both sexes being infected. As clinical studies of these cases were made, it is interesting to note that no uniformly bad effects can be attributed to the very heavy infections seen. At times gastro-intestinal disturbances were noted, even entero-colitis. The elaborate train of nervous symptoms usually described were as a rule conspicuous by their absence. There are at least two serious aspects to ascariasis; (1) the tendency to upward wandering, causing vomiting of worms, penetration into the gall ducts and even into the liver, perforation of the bowel, generally through a preexisting ulcer, etc.; and (2) intestinal obstruction. In a small number of cases, however, the worm undoubtedly acts as a local irritant even to the extreme of simulating appendicitis or gastro-entero-colitis, and of indirectly producing profound nervous disturbances, or a cachectic condition.

Treatment.—We are accustomed to give 1 centigram of santonin for each year to 25 years of age, combined with an appropriate dose of calomel. This is administered at bedtime in two doses, 2 hours apart, after a day of fasting, and followed next morning by a dose of castor oil. Great caution is recommended in using this drug as dangerous, if not fatal, accidents occur in young children. Usually it is a safe remedy, but somewhat undependable. Neither *Trichuris trichiura* nor *Strongyloides stercoralis* seems ordinarily to be definite factors in the causation of disease. The Institute of Porto Rico finds the first a very common parasite in man. Said to cause colitis, marked anemia, and nervous symptoms, we have never been able to incriminate this worm. It is difficult to expel, but thymol is sometimes successful. Strongyloides stercoralis is less common in Porto Rico but not rare. It does not seem to cause diarrhea as reported by other authors, but infections may not have been heavy enough. It is especially found in brickyard workers, miners' tunnel workers and barefooted agricultural laborers as its mode of infection is similar to that of uncinariasis. The embryo, not the ovum, is what is usually found in the feces.

Cestodes.—Tænia saginata frequently, and T. solium much less commonly are found in these who eat insufficiently cooked, or even raw beef or pork, such as butchers, cooks, and sausage makers. These parasites are ubiquitous, but are rare where meat inspection and a proper disposal of excrement are exacted. Among Porto Rican agricultural laborers, where meat is rarely consumed and always well cooked, our Institute of Tropical Medicine, in nearly 10,000 nematode infections studied in 1913, failed to find one case of teniasis. Tænia saginata, 4 to 18 meters long, is a white flat worm with an unarmed head and about 1000 hermaphroditic segments (proglottides) each containing a uterus with from 25 to 35 lateral branches. Tænia solium,

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2 to 3 meters long, has a double row of hooks upon its rostellum and each proglottis contains a uterus with from 7 to 10 lateral branches. The ova, ingested by cattle (T. saginata) and by hogs (T. solium), develop into embryos which finally encyst in their flesh, forming cysticercus bovis and cysticercus cellulosæ respectively. The habitat of the mature worms is man's intestine which they never perforate, and from which they rarely wander. There is little doubt but that a tape-worm infection may be and frequently is compatible with apparent health but in the feeble symptoms are more apt to arise, usually those of a gastro-intestinal dyspepsia often with boulimia and obscure nervous manifestations generally limited to nasal and anal pruritus, dry cough, vertigo and cardiac palpitation. Occasionally the cysticercus stages of T. solium is passed in the tissues of man, and as these cysts, 0.006 to 0.020 mm. long, at times invade the eye and brain the prognosis is extremely grave. Whatever tenifuge be selected the patient should (1) take the drug on an empty stomach, and (2) follow up the last dose within a half hour by a vigorous purge to expel the stunned worm. Three drugs are worthy of special mention: (1) pelletierin tannate, preëminently effective but dangerous, (2) male fern, given with calomel in ½-gram capsules of the etheral extract, two every 10 minutes to 6 or 8 grams, and, (3) thymol as recommended for uncinariasis. Where toxicity is feared pumpkin seed, kousso or the expressed juice of cocoanut meat may be employed. The prophylaxis consists in treatment of all infected, strict national and international meat inspection, and proper disposal of human feces.

TREMATODES

Schistomiasis.—Caused by Schistosoma hematobium, whose terminal spined ova produces a serious inflammation of the urinary tract with bleeding papillomatous granulation tissue, chiefly of the bladder wall, notably among the Fellaheen of the lower Nile who work in the fields up to their knees in water; by S. japonicum, encountered in the far East and causing grave hepatic and intestinal lesions often leading to cachexia and death; and by S. mansoni, the parasite responsible for American Schistosomiasis. In the latter disease, common in the West Indies and South America the histopathology is similar to that caused by S. hematobium but the lesions are found in the colon and rectum. The ova are all lateral spined and contain a miracidium which liberated in water infect through the skin, according to Looss, without the intervention of an intermediate host. In the series of 10,140 cases of the Institute of Tropical Medicine of Porto Rico, 1913, over 200 were found to be infected, very few of whom showed definite symptoms save a suspicious epigastric pain. These patients all gave a history of having bathed in a river suspected of being contaminated. Heavier infections cause "Schistosoma dysentery," a proctitis roughly simulating chronic

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dysentery, but with a tendency to prolapse of the rectum. The prognosis is generally favorable in light infections, but treatment is futile. The prophylaxis is generally complete if one avoids bathing or wading in infected streams and the ingestion of unboiled water or contaminated fruits and vegetables. In Porto Rico we are not prepared to consider Looss' theory of skin infection as proven.

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CHAPTER III

COMPRESSED-AIR ILLNESS

BY SEWARD ERDMAN, M. D., New York City, N. Y.

Synonyms.—"Caisson disease," "divers' palsy," "bends," "screws," "courbatures," "aeropathy," "aeræmia."

Definition.—An occupational disease occurring among divers, caisson workers, subaqueous tunnel workers, etc., following exposure to compressed air, the symptoms of which arise only after the too rapid removal of pressure (decompression), which liberates in the supersaturated body fluids and tissues bubbles of gas.

The symptoms are usually of sudden onset within from 15 minutes to a few hours after decompression, and include pains, paralyses, dyspnœa, and even circulatory failure and death.

There is a strong tendency to early and spontaneous recovery in the majority of cases.

Historical.—Descriptions of compressed-air illness date back only 75 years and it is essentially a disease of modern civilization.

In 1839 Triger, a French engineer, who elaborated the first practical caisson and employed it in mining coal under the River Loire, describes the occurrence of pains in the extremities of certain of his caisson workers, and these observations constitute the earliest authentic recognition of compressed-air illness.

However, from the times of Alexander the Great and Aristotle onward through the centuries, crude diving bells and helmets had been experimented with, and in 1665 salvage from the sunken Spanish Armada was accomplished at the depth of 46 ft.

In 1717 Halley devised a diving bell which could be used at a depth of 60 ft.

A century later Siebe, in 1819, devised the first diving helmet supplied with air from an air pump, and this diving dress which he perfected in 1837 is the prototype of those now in use.

The principle of the caisson was patented by Cochrane in 1830 but first made a working device by Triger.

Doubtless in the earlier experiments technical difficulties prevented exposure to sufficient pressures for a sufficient time to give rise to symptoms of compressed-air illness.

Scientific investigation of caisson disease was earliest undertaken by Pol and Wattelle[1] in 1854, and although the mechanical pressure theory which

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they put forward has since been entirely rejected, nevertheless many of their observations and conclusions have remained uncontroverted.

In 1857 F. Hoppe-Seyler, repeating the experiments, which Boyle had begun in 1670, upon animals subjected to rarefied atmosphere by use of the vacuum pump, confirmed the fact of the liberation of bubbles of gas in the body fluids.

By analogy, Hoppe-Seyler propounded the correct theory of the causation of caisson disease.

P. Bert[2] in 1878, by the first animal experiments with compressed air, proved conclusively the truth of Hoppe-Seyler's theory, and his work is still regarded as a classic in the literature of compressed-air illness.

Most valuable experimental work and contributions to the study of this disease are to be attributed to such men as Heller, Mager, von Schrötter,[6] Silberstern,[14] Hill,[12] Greenwood, Macleod,[16] Haldane, Boycott, Damant and to numerous others.

The building of the East River tunnels from New York to Long Island City for the Pennsylvania R. R. in 1906–1909 afforded material for monographs by F. L. Keays,[8] H. Japp,[13] McWhorter[10] and the writer,[9–15] dealing chiefly with the practical and clinical aspects of the illness.

Frequency.—Occurrence of this disease postulates the use of compressed air, and consequently its frequency has greatly increased with the development of submarine construction work and diving.

Caisson work for the Kehl bridge over the Rhine in 1859 was one of the earlier large ventures. The St. Louis bridge, 1870, and the Brooklyn bridge, 1873, also the Danube bridge, 1890, afforded opportunities for the further study of caisson disease.

However, river-tunnel construction employs so many more men in compressed air than does caisson work that we turn to those larger enterprises to furnish the massive statistics for a clinical study.

The East River tunnels employed over 10,000 men during their construction, at an average working pressure of +32 lb. with a maximum of +42 lb.

The writer[9] has reported on 1 year's work, and F. L. Keays,[8] the medical director, has ably analyzed the total statistics which embrace 3692 cases of illness among the 10,000 men, or 36.9 per cent. But I feel certain that if every trivial case of "bends" had been recorded, the percentage would have to be doubled; indeed I doubt that even 10 per cent. escaped unscathed at all times.

At the Eider bridge in 1885, with a maximum pressure of +35 lb., there occurred 380 cases among 140 workers, or 270 per cent.

There were reported 320 cases among 675 men who worked at the Danube bridge in 1890 at a maximum pressure of +32½ lb., and this represents 47.4 per cent.

But as the possibility of the development of symptoms is present every

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time the pressure is removed, comparison with the number of man shifts would be a more accurate method of estimating the frequency of the disease.

Thus, at the East River tunnels there were 557,000 decompressions, and the 3692 cases of illness would give an incidence of only 0.663 per cent.

The British Admiralty Committee on Deep Sea Diving reported 60,000 decompressions with 577 cases, or 0.96 per cent.

General Discussion of the Nature of Compressed-air Illness, Based on the Accepted Gas-bubble Theory.—When a man or an animal is exposed to compressed air, the blood, by diffusion from the alveoli, and later all the body fluids and tissues become supersaturated with the nitrogen of the atmosphere to a degree dependent upon three factors: (1) the amount of pressure (see Dalton's law); (2) the length of exposure to the pressure; (3) the vascularity and the absorptive ability of the individual tissues.

Upon decompression the excess nitrogen (see below) tends always to flow off from the supersaturated tissues, by means of the circulating blood, into the alveoli and expired air where the pressure is lower.

Such a gradual and orderly desaturation of the body tissues would occur under favorable conditions without the ebullition of any bubbles of free gas, and no symptoms would then result.

As will be shown, however, the desaturation of the tissues always lags behind the lowering of pressure, as decompression is commonly practised, and there is left a residuum of gas in the organism.[11]

Therefore it follows that there is always the possibility that too rapid removal of pressure may result in the freeing of nitrogen in the form of bubbles in the tissues and body fluids.

Probably if the bubbles be small in size and amount and occur in indifferent tissues, the circulation will get rid of them by way of the lungs and still no symptoms may arise.

When free bubbles of gas by their expansion or coalescence form gas emboli in the circulation, or inflict direct mechanical injury upon delicate nervous tissue by expansion of the bubbles in accordance with Boyle's law, then symptoms may and do result.

Fat, fluids, and nerve tissue are especially affected; consequently the irritation of peripheral nerves, or injury to the central nervous system by interference with its blood supply and by pressure of expanding bubbles, will give rise to many of the protean symptoms which characterize this malady, and in severe cases the resultant softening or hemorrhages may cause a true myelitis.

Compressed-air illness then is the direct result not of the increased pressure, but of decompression which has been so rapid that bubbles of free nitrogen have been liberated in the organism.

Hence the symptoms never occur under compression, however long continued, but only as the result of, and after, decompression.

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Such in brief is the gas-bubble theory which has been indisputably established as the correct and only explanation of compressed-air illness.

A large proportion of all autopsies performed within 24 hours after death in the rapidly fatal cases, whether among men or in animal experiments, show visible bubbles of gas in the veins and right heart and often in other tissues.

Theories.—Other theories have been advanced and tenaciously held, but they must all fall to the ground if for no other reason than that they do not even attempt to account for the formation of a single bubble of gas.

1. The *mechanical pressure* theory propounded by Pol and Wattelle[1] in 1854, and held by A. H. Smith and others, is based upon false conceptions of the effect of compression upon the vascular system, and it fails to explain the pathological findings.

2. The "effects of *cold* and *fatigue*," whose adherents include Jaminet,[3] Tinc and others, has truth in it only to the extent that fatigue and cold are minor contributing factors.

3. The *carbon dioxide poisoning* theory of Snell[4] does not explain the phenomena of gas-bubble formation; also its symptoms would occur during exposure to pressure, which is never true of compressed-air illness.

Furthermore Hill[12] and Greenwood, also Haldane and Priestley, have proven that the absolute partial pressure of CO_2 in the alveoli is regulated by the rate of respiration and remains practically the same (5.6 per cent.) under compression even up to +6 atmospheres, and consequently no more is taken into the blood under pressure. Haldane[7] states that even when the air in a caisson or diving helmet is so vitiated as to contain 3 per cent. CO_2 pressure it will not affect the man in his work, although it would increase his respirations; if it contain more than 4 per cent. CO_2 pressure, the resulting dyspnœa would interfere with hard work, and only when the CO_2 pressure is above 10 per cent. will unconsciousness from suffocation supervene.

4. The *oxygen poisoning* theory of Lorrain Smith is based on the fact that the breathing of very high pressures of oxygen, at from +7 to +15 atmospheres during long exposures, produces congestion and inflammation of the lungs.

As an explanation of compressed-air illness, this theory is of course quite inadequate.

ETIOLOGY—PREDISPOSING FACTORS

1. **Degree of Pressure.**—Other things being equal, the number of cases of illness increases directly with the degree of pressure.

Only a very few minor cases of pain occurred at pressures up to +20 lb. One case of transient hemiplegia with aphasia after 8 hours at +15 lb. was reported by Keays,[8] although the hemiplegic case depends solely on the patient's own statement for he was not seen by a physician.

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Haldane thinks that bubbles will not form even after the most rapid decompression from pressures up to +15 lb., and adds that symptoms begin with +20 lb., are rare up to +30 lb., but are serious in frequency above +30 lb.

Bert,[2] 1878, was told of one fatal case after exposure to +21 lb., but there seems to be reason for doubting the correctness of this report.

Haldane never heard of a fatal case below +29 lb.

At the East River tunnels the lowest fatal case occurred at +28 lb.

From a wide practical experience, Mr. Japp[13] considers that pressures up to +27 lb. may be regarded as free from the risk of serious developments.

However, it is clear that every additional pound above +30 lb. carries with it increasing risk.

2. **Length of Exposure.**—The degree of saturation of the organism, is for any given pressure, a matter of time. Were it merely a question of the saturation of the blood, this might take place in 1 minute, if we premise, as is probably true, that the diffusion of gases from the alveoli to the blood occurs instantaneously and that the complete circulation of the blood takes place in 1 minute (Plesch says 55 seconds during rest but only 4½ seconds during hard work).

However, the saturation of the body fluids and tissues takes place only gradually from the blood, and those tissues which are deficient in blood supply will require more time for saturation.

Again we find that certain tissues are better solvents for nitrogen than others. Thus, Vernon[12] estimates that fat can dissolve five times more nitrogen than the blood and non-fatty tissues, by bulk. Hence fat, which makes up from 15 to 20 per cent. of the body weight and is little vascular, will saturate slowly and consequently desaturate slowly.

Therefore as a unit in saturation of the organism containing so much fat, the blood has an effective bulk of only $\frac{1}{35}$, although in reality it is from $\frac{1}{20}$ to $\frac{1}{13}$ of the body weight.

Attempts have been made to calculate the rate of saturation of the body tissues on the basis that $\frac{1}{35}$ saturation occurs in 1 minute and $\frac{1}{35}$ of $\frac{34}{35}$ in the second minute, etc. Thus Haldane[7] and Boycott estimate that 22 per cent. saturation will occur in 5 minutes, 50 per cent. saturation in about 25 minutes and almost complete saturation in 90 minutes, but the slowest parts might require 5–9 hours for complete saturation.

Turning from the theoretical consideration of this question to the results of practical work in compressed air, Keays[8] found that among 2719 men exposed to +31 lb. pressure for 1½ hours on a preliminary test and therefore not working, only three cases (0.18 per cent.) of illness developed, whereas among 2000 men working for 1½ hours at +32 lb. there were seven cases or 0.35 per cent. (In this instance the length of exposure was the same, but the effect of work in hastening saturation is illustrated.) After two 3-hour shifts at 31.7 lb. 1.46 per cent. developed symptoms, in 3360

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man shifts, whereas in 23,760 man shifts (two shifts of 4 hours each) there were 216 cases or only 0.90 per cent.

From these statistics Keays concludes that saturation is not complete after 1½ hours, although it is more nearly so if the men are working than if they are resting; that after 3 hours' work saturation is practically complete and longer exposures, *e.g.*, of 4 hours, did not increase the percentage of cases of illness. In fact the percentage after the 4-hour shifts was smaller than after the 3-hour shifts, which apparent discrepancy I would attribute to the fact that the longer interval between shifts (4 hours for the 4-hour shifts and 3 hours for the 3-hour shifts) enabled the 4-hour men to become more completely desaturated as well as rested before going down to their second shift.

No less an authority in practical engineering than Mr. E. W. Moir[12] states that he "is confident that a limitation of the exposure to even 3 hours distinctly diminishes the risk of bends as compared with 6 or 8 hours' exposure.

However, we may conclude from the statistics cited above that for practical purposes saturation occurs after 3 hours' work under pressure, for the average man.

3. **The Personal Equation.**—A. *Efficiency of the circulation* would epitomize the qualifications for good air workers, and any condition which tends to lower the tone and elasticity of the blood-vessels or to cause passive congestion will in so far retard desaturation and predispose to the collection and coalescence of gas bubbles in the circulation. Hence we note as predisposing factors debilitation from disease or malnutrition, from alcoholic and other excesses; constipation; fatigue; the effects of cold and the lack of exercise during decompression and afterward.

B. Organic diseases, especially arteriosclerosis, heart lesions, respiratory diseases, interfere with rapid desaturation. Anæmia, œdema, status lymphaticus have all been reported in some of the fatal cases, and naturally such diseases should prohibit work in compressed air.

C. Age.—Men from 18 to 30 years of age are the most desirable for pressure work, because of their efficient vascular and general condition. Beyond 30 years the risk of serious illness increases, and with few exceptions men over 40 years of age should be excluded.

D. Fat men are notoriously ill-fitted for work in high pressures, a practical observation which is fully explained by the slow desaturation of fat, and substantiated by the animal experiments of Boycott and Damant.

E. Fatigue may be conceived as playing a rôle in the causation, by impairing the efficiency of the circulation; indeed, Keays[8] holds it responsible for the fact that for the same men at the same pressures there was an increase of cases of illness after the second 3-hour shift (0.72 per cent.) when compared with the first 3-hour shift (0.35 per cent.). However, I would note another possible factor here, namely, the incomplete desaturation during the 3-hour (often 2½-hour) interval between shifts.

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4. **Atmospheric Conditions.**—Above ground have been thought by some men to affect the incidence of illness, but we were not able to establish any such connection at the East River tunnels, where we kept a daily record of the temperature, barometer readings, humidity and direction of the wind.

The humidity in the tunnels was always nearly at saturation, never under 90 per cent. (McWhorter).[10]

5. **Noxious Gases.**—The possible presence of injurious quantities of carbon dioxide, of carbon monoxide, of methane, of sulphuretted hydrogen, or of nitric oxide in a tunnel or caisson might call for remedy in the way of better ventilation; but even supposing their presence in excess, either from the compressors, or from blasting, or from any cause whatsoever, the resultant symptoms would be due to the exciting cause and would develop while the men were under pressure, and although conceivably they might be predisposing factors, they are never essential factors in the etiology of compressed-air illness.

Carbon Dioxide.—At the East River tunnels, McWhorter's daily analyses of the tunnel air showed such excellent ventilation that the maximum carbon dioxide reading was 0.1 per cent. (the equivalent of 0.3 per cent. at 3 atmospheres); the minimum was 0.045 per cent., and the carbon dioxide variations had no effect upon the percentage of illness.

Carbon Monoxide.—Haldane states "that anything over 0.15 per cent. of carbon monoxide must be regarded as distinctly dangerous, and probably anything over 0.03 per cent. would in time produce symptoms distinctly felt on exertion."

Immediately after blasting in our tunnels, samples of air from the headings showed a maximum of 0.045 per cent. CO, but this was rapidly removed by ventilation; further, McWhorter says that although some of the men complained of headache after the blasting, he does not regard this as a symptom of carbon monoxide poisoning.

Methane was never found and nitric oxide and sulphuretted hydrogen only occasionally and in most minute quantities.

Exciting Cause.—The immediate and exciting cause of compressed-air illness is too rapid decompression. (See Treatment, for details.)

Immunity.—This is purely a relative matter and probably no absolute immunity exists, but the best-adapted men may escape symptoms, at least for a long time, and rarely one encounters old and experienced workers who have never had a twinge of the "bends."

Pathology.—Bubbles of gas, set free in the tissues and fluids of the body, give rise to the pathological lesions found at autopsy, as well as to the transient symptoms which characterize the non-fatal cases.

The pathognomonic and most striking lesion is the presence of gas bubbles (gas emboli) in the veins of the systemic and portal system, in the right heart, and in the pulmonary and coronary vessels. These bubbles are found so constantly in animal experimentation and in the majority of autop-

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sies upon men in the rapidly fatal cases (provided the autopsy be performed within 24 hours after death) that they constitute the basis of our understanding of this disease.

Death in many cases is the result of distention of the right heart with gas, or of pulmonary air embolism.

Nature of the Gas.—Analyses of the gas drawn from the right side of the heart of animals killed after rapid decompression from high pressures are represented by the following:

	Nitrogen, per cent.	Oxygen, per cent.	Carbon dioxide, per cent.
Paul Bert,[2] 1878:			
1. Dog	82.8	2.0	15.2
2. Cat	84.1	None	15.9
Von Schrötter:[6]			
1. Dog	79.98	15.31	4.71
2. Dog	80.37	7.18	12.45
McWhorter[10] and Erdman:[16]			
1. Tunnel worker*	80.0	None	20.0

How then are we to explain these analyses if we maintain that nitrogen is the sole factor in supersaturation? Haldane states that the amount of oxygen taken by the blood under conditions of pressure adds but little to the total oxygen of arterial blood and in any event readily combines with the tissues, and for the reasons stated under "the carbon dioxide theory" we have seen that no more than the normal amount of CO_2 is taken by the blood.

Why do not the analyses reveal nitrogen alone? Hill[12] explains this by saying that "when nitrogen is set free as bubbles, these act as a vacuum for CO_2 and oxygen, and some of these gases are set free out of chemical combination with the blood; and as the oxygen is mostly used up by the tissues of the dying animal, we find the chief gas, besides nitrogen, is CO_2." (Witness the analysis from the human heart, above recorded.)

The *nervous system* may or may not show lesions at autopsy, although irritation of the peripheral nerves or spinal cord, by bubbles, gives rise to nearly 90 per cent. of the symptoms of the disease.

* So far as I can find, this latter is the only recorded analysis of gas from the human heart after death from compressed-air illness.

The autopsy was performed, about 14 hours after death, upon a tunnel worker who died Nov. 19, 1907, 1 hour after decompression, having been found in a state of collapse and unconsciousness near the works. The man had worked 8 hours in a pressure of +30 lb. and had left the tunnel by a 17-minute uniform decompression. The right heart was found to be ballooned with gas and some of this (3.1 cc.) was aspirated into a specially devised collector after flooding the thorax with water and excluding all air from the collector. The analysis was made by Dr. Metzger of Columbia University.

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Fat acts as a reservoir for nitrogen, and Vernon estimates that the spinal cord and the peripheral nerves contain 20 per cent. fat.

Especially in cases of paralysis, autopsy may show capillary hemorrhages, or areas of softening due to embolism, in the spinal cord, or even laceration of nerve cells by the expanding bubbles. In persistent paraplegias, a true "caisson myelitis" is found.

The lower dorsal and the upper lumbar regions are most often affected because of their meager blood supply in comparison with the functionally more active segments. Gray matter has a richer blood supply than white matter and so more often escapes.

Gas bubbles or resultant capillary hemorrhages and tissue changes may be found in the liver, spleen and other organs; indeed bubbles may occur in nearly all the body tissues and more detailed mention will be found in the symptomatology given below.

Phenomena of Compression.[15]—Certain interesting physical and mechanical phenomena are noted during the process of compression:

1. The rising pressure forces inward the ear-drum membrane, causing discomfort, acute pain, or even rupturing this structure, unless the pressure be equalized by admitting air through the Eustachian tube, by Valsalva's method or an equivalent, *e.g.*, swallowing. This pressure on the ear-drum membrane is most marked with the first few pounds and so it is possible to rupture the membrane with +5 to +10 lb. pressure, but this cannot be considered a symptom of compressed-air illness.

2. Slight rise in body temperature and sweating occur, due to the heat generated by the compression of the air.

3. The denser air offers a slight resistance to expiration and phonation. Whispering becomes impossible, and the lessened amplitude of vibration renders whistling difficult or impossible, and in such an attempt the non-vibrating lips give a sensation of slight numbness.

4. The voice loses its natural quality and sounds intensely nasal.

5. A sense of exhilaration and an ease of movement are experienced, and it is claimed that compressed-air work increases the appetite.

Other phenomena of compression have been reported, but are now generally discredited; thus:

Pallor of the skin and mucous membranes does not occur unless it be from nervousness on the part of the man entering compressed air for the first time.

There is no lessening of the volume of the pulse; no constant alteration in the blood pressure; no constant change in the rate either of the pulse or respirations.

The superficial veins do not collapse; the capillary circulation in a frog's foot continues unchanged (Hill and others).

There is no gross change in the urinary or sweat secretions. Hearing is not affected.

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Phenomena of decompression include chilling of the body, due to the falling temperature in the air lock, and one may at times note a crackling sound in the ears as the air escapes through the Eustachian tubes. With rapid decompression the moisture in the atmosphere condenses and the lock fills with a dense fog.

Symptoms.—The symptoms of compressed-air illness occur only as a result of, and after completion of, decompression; and no symptoms ever develop while a man remains in the compressed air, no matter what the degree of pressure nor the length of exposure.

A. *Onset.*—The time of onset is from within a few minutes up to a few hours after decompression. At the East River tunnels about 50 per cent. of the 3692 cases developed within 30 minutes, and 95 per cent. within 3 hours; however, 1 per cent. were delayed over 6 hours and of these, four isolated and somewhat doubtful cases were said to have occurred between 15 and 23 hours after decompression (Keays).

Bert says that the bubbles do not form all at once but continue to form for some time after decompression.

The onset of symptoms is often dramatic in its suddenness. A man in all the vigor of health and strength descends to his daily task in the river tunnel; enters the compressed air; works his regular shift of 3 or 4 hours; comes out through the air lock by the ordinary method of decompression; ascends to the street and starts for home, feeling perfectly well.

Fifteen minutes later, without the slightest warning, he is attacked with intense boring pains in his legs and abdomen, or he staggers and falls helpless to the ground, paralyzed from his waist down. Fortunate he is if efficient treatment be near at hand, and by recompression in the medical air lock, his pains vanish, his paralysis disappears, and on the morrow he returns to his work as usual.

B. *Chief Symptoms in Order of Frequency:*

1. Pains in the extremities or abdomen occur in 88 per cent. of all cases.
2. Vertigo, in about 5 per cent.
3. Cerebrospinal cases, in 2.16 per cent.
4. Dyspnœa, or "chokes," in over 1.5 per cent.
5. Prostration of moderate degree, with pains, in 1.25 per cent.
6. Collapse with unconsciousness, in 0.46 per cent.

C. *Classification of Acute Symptoms.*—The symptoms of compressed-air illness will vary with the anatomical part affected, with the amount of gas set free, and with the consequent degree of disturbance of function; accordingly we will group the symptoms with reference to the structures affected.

1. **Nervous System.**—(*a*) *Peripheral Nerves.*—*Pain* is by far the commonest symptom, occurring in over 88 per cent. of all cases, either as the only symptom or associated with others. Its most frequent (70 per cent.) site is in the lower extremities in the region of the knees, forcing the victim into an attitude which gives rise to the popular term "bends," or "courbatures."

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Next in frequency are pains in the upper extremities, about the elbows and shoulders in 30 per cent. Pains in the abdomen occurred in about 5 per cent. of the cases, but were of greater significance than were pains in the extremities in that they more often preceded or accompanied cases of severe prostration or paralysis.

Pain occurring in the extremities or abdomen in over 88 per cent. of all cases, without other evidence of spinal-cord irritation, except in a little over 2 per cent., must admittedly be due to peripheral irritation of nerves and nerve terminals by bubbles of gas in the nerve sheath, in fascial tissues, beneath the periosteum or in bone marrow, etc.

Numbness, anæsthesia, and paræsthesia are doubtless capable of the same explanation.

One form of paræsthesia, called by the workmen "itch," is quite common, and distressing while it lasts, but is never of serious import. This may be explained, as Hill does, as due to bubbles in the subcutaneous areolar tissue, but in my experience it is no more common in fat men than in thin men;[15] it occurs especially when sweating has been less free, and it is usually at once relieved by a hot bath or sweating, all of which make it more probable that it is caused by expanding bubbles in the sweat glands.

The "itch" or "puces" has been attributed to irritation of the posterior nerve roots by bubbles in the spinal fluid.

(*b*) *Central Nervous System.*—Brain: unconsciousness, stupor, and collapse may be due to cerebral gas embolism or the resulting œdema, or are secondary to circulatory failure due to coronary or pulmonary embolism.

Cerebral gas embolism may also cause temporary aphasia, incoherence of speech, ataxia, vomiting, vertigo, headache; or such signs as hemiplegia, monoplegia, convulsions, nystagmus, and tongue deviations, depending upon what area is affected.

Actual tearing of nerve tissue and hemorrhage may arise from the expansion of bubbles of gas, resulting in organic lesions.

Vertigo, or, in the parlance of the workers, "staggers," was met with in over 5 per cent., and is explainable by the formation of bubbles in the labyrinth of the internal ear, or when accompanied by nausea and vomiting it may be due to cerebellar gas embolism.

Spinal Cord: Affections of the spinal cord include paraplegia, monoplegia, loss of bladder and rectal control, partial paralyses, spasticity, exaggerated, diminished, or absent reflexes, anæsthesia, anæsthesia dolorosa, etc.; and with hemorrhage into the cord the symptoms may become permanent and a true myelitis may develop.

Frequently, however, there is only a temporary weakness and numbness of the legs, with or without retention of urine.

2. **Special Senses.**—(*a*) *Eye.*—Transient blindness, diplopia and nystagmus may occur.

(*b*) *Ear.*—Perforation of the membrani tympani due to compression is

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not properly a symptom of the illness. Deafness and vertigo or Ménière's disease may result from the formation of bubbles in the labyrinth.

3. **Vascular System.**—The phenomena of gas emboli in the blood-vessels has been described, and undoubtedly the cases of extreme prostration and collapse and many of the fatalities are due to the collection of gas in the right side of the heart.

A bluish "mottling" of the surface of the chest or abdomen, encountered in some of the more severe cases, is due to embolism of the superficial veins.

Lymphatic obstruction by gas emboli explains the several cases of localized œdema which I have observed.

4. **Respiratory System.**—A form of dyspnœa, called "chokes," of a type resembling an asthmatic attack, may well be due to multiple small emboli in the pulmonary vessels; for were it of central nervous origin, it would probably be attended by other serious nervous symptoms, which is not the case.

In certain fatal cases there has been found œdema of the lungs, and at times an interstitial emphysema.

5. **Gastrointestinal symptoms** further than the epigastric pains, the nausea and vomiting, above described, are not encountered.

6. **Internal organs,** such as the liver, spleen, kidneys, do not give any acute signs, although at autopsy they may be found affected.

7. **The sexual apparatus** is affected only as a result of spinal-cord injury, which may give rise to priapism, etc.

8. **The Bones, Joints and Periosteum.**—In one case necrosis of the femur resulted apparently from thrombosis of the medullary artery. The deep-seated pains so frequently referred to the long bones of the extremities and to the joints may be due either to collections of expanding bubbles under the fascial planes or under the periosteum; or, as Hill suggests, in the yellow marrow, which structure is rich in fat and deficient in circulation.

9. **The Skin and Subcutaneous Tissues.**—The symptoms of "itch" and the "mottling" of the skin have been described above. Subcutaneous emphysema, without trauma, is met with in a few cases during life, and frequently in autopsies, especially along the course of the large vein of the extremities. Its presence as an accompaniment of contusions and punctured wounds I have several times demonstrated, and it is doubtless due to the setting free of gas from the extravasated blood; for it is hardly likely that such an amount of compressed air could enter through the puncture.

10. **Localized collections of gas,** aside from the small emboli scattered through the body, include the large collections found in the right side of the heart at autopsy (case cited above). At one autopsy which I witnessed there was a collection of about 5 cc. of gas beneath[9] the mucosa of the jejunum. In two other cases I succeeded in aspirating about 1 cc. of gas from beneath[9] the periosteum of the tibia; its presence was evidenced by a soft cushion-like swelling in this area.

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11. **Miscellany.**—In general, the wounds bleed as freely and heal as readily in the compressed air as in the normal atmosphere.

Complications.—Perforation of the membrani tympani may be called a complication; also infections of the middle ear and of the sinuses which communicate with the nose are favored by the efforts of the men to force open the Eustachian tubes, and these factors account for the infrequent cases of epistaxis in caisson or tunnel work.

In diving, however, bursting of the air tube has a cupping effect and may cause epistaxis, ecchymoses and rupture of the membrana tympani in an outward direction.

Cystitis, pyelonephritis, myelitis and meningitis may be later results of spinal-cord injury.

Symptoms: Chronic.—(*a*) *Myelitis.*—At the East River tunnels, exclusive of the fatal cases which died within 8 days, there were 10 cases of permanent paralysis, or 0.3 per cent. of the total cases. Of the 10, four died within 5 months from complications, three were lost track of, and the remaining three, at last reports were still afflicted with spastic paraplegia.

(*b*) *Permanent Ear Troubles.*—I knew of but three cases (in the 3692), of ruptured membrana tympani and with persistent impairment of hearing; one of these developed an obstinate otitis media purulenta.

As has been said above, these cases are perhaps complications of work in compressed air but are not symptoms of compressed-air illness; rather were they accidents of compression.

However, cases of labyrinthine deafness are reported as setting in after decompression, and due to bubbles in the labyrinth; such cases of which I saw no permanent ones, would be properly classed as symptoms of this disease.

Ménière's symptom-complex is of similar origin and I knew of one persistent case.

That many and vague symptoms in after life are attributed to the effects of compressed-air illness, which have, however, no actual connection therewith, is a fact to which the testimony submitted in certain damage suits bears eloquent testimony.

Bassoe[18] examined 161 men at a period of months or in some cases, years after they had worked in compressed air, and he reports 16 cases of demonstrable permanent injury, or 10 per cent., and ear troubles in over 60 per cent.

His grouping is as follows:

Class 1. Caisson myelitis.
- (*a*) Trophic osteo-arthropathy due to cord lesion (cases, three).
- (*b*) Very limited persistent cord lesions (cases, two).
- (*c*) Caisson myelitis more or less extensive (cases, three).

Class 2. Permanent joint disease (arthritis deformans) cases, seven.

Class 3. Ear affections. 67 men complained of impaired hearing.
33 complained of dizziness.

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Certainly from our experience at the East River tunnels in the 3692 cases we consider Bassoe's statistics quite amazing. In a number of his cases, we feel that the very essential relation of cause to effect is not sufficiently well established.

In a word we may state that as a result of Keays' analysis of the East River tunnel cases, the total of permanent injury cases does not exceed 1.0 per cent. even when we include all fatal cases and all persistent paralyses and permanent ear affections.

Mortality.—Except for the doubtful case related by Bert of a man who died after exposure to + 21.1 lb. (?), I can find no record of fatalities occurring under +28 lb.

An idea of the great variation in mortality at different works may be obtained from the following statistics:

(*a*) Mortality in relation to pressure and cases of illness. We believe that with modern methods of decompression and treatment the mortality should never amount to even 0.5 per cent. of the cases of illness.

	Max. pressure, lb.	Cases	Deaths	Percentage
1839, Douchy mines	+37.5	63	2	3.2
1870, St. Louis bridge	+50.0	129	14	10.8
1873, Brooklyn bridge	+34.0	110	3	2.72
1885, Hudson tunnel	+35.0	50 workers	9	18.0 plus
1890, Danube bridge	+32.5	154	4	2.6
1895, Danube bridge	+37.5	320	2	0.625
1906, Amsterdam	+30.0	228	none	....
1909, East River tunnel	+32–42	3692	20	0.54

In comparing the above figures, note that only the maximum pressure employed at any period of construction is given, except in the case of the East River tunnels where the average for the whole work was +32 lb. and the maximum was +42 lb.

At Amsterdam where there were no deaths it will be seen that the pressure at no time exceeded +30 lb.; and as has been stated it is every additional pound above +30 which especially enhances the danger of serious cases.

(*b*) Mortality according to number of workers employed:

	Men	Deaths	Percentage
Hudson tunnel	50	9	18.0
East River tunnel	10,000	20	0.2

(*c*) Mortality, according to decompressions:

	Decompressions	Deaths	Percentage
Admiralty Com. Report	60,000	4	0.0066
East River tunnels	557,000	20	0.0035

Types of Fatal Cases.—The 20 fatal cases at the East River tunnels were either rapidly fatal (within a few hours or a few days), or delayed for weeks or months.

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A. Rapidly fatal cases.

1.	Pains accompanied by marked prostration,	6 cases.
2.	Collapse and early unconsciousness,	9 cases.
3.	Paraplegia, with coma later,	1 case.

B. Delayed Deaths.—These were due to complications in cases of myelitis; *e.g.*, cystitis, pyelitis, bed-sores, etc.; in one case death occurred on the tenth day from an ascending paralysis. Only one of the twenty lived as long as 5 months.

Diagnosis.—The diagnosis is made from the history which will consist in the onset of symptoms within from a few minutes to a few hours after decompression from compressed air. Without such a history, the air worker who falls unconscious or hemiplegic upon the street, may well baffle the best diagnostician.

Prognosis.—Simple pains, or "bends," limited to the extremities are rarely of serious import, and occurred in 88 per cent. of all cases; they are often transient and shifting and usually yield at once to recompression. Pain in the abdomen often precedes severe symptoms.

The Admiralty[12] Committee considers "bends" to be the sign of saturation of very slow parts, which will be slow to desaturate, and therefore the "bends" are most difficult of entire elimination by any practical method of decompression; further they occur independent of the amount of fat in the subject.

The more severe symptoms of dyspnœa, mottling, paralyses, collapse and unconsciousness, bespeak inadequate de-saturation of the quick parts, and indicate much gas in the blood and are accordingly of serious portent.

Of the 34 cases of paralysis, 23 recovered (19 of these after one recompression).

Of 17 cases with early lapse into unconsciousness, 9 were fatal and represented 45 per cent. of the total mortality.

To recapitulate we may state that many cases of the "bends" will subside in at most a few days even without recompression. About 90 per cent. of all cases were entirely relieved by treatment, and 99 per cent. were in large part relieved by recompression.

The fatal cases and the permanent injuries, taken together, amounted to only 1 per cent. of the 3692 cases.

Treatment

1. Prophylactic.
   - A. Choice of men.
   - B. Hygiene of the workers.
   - C. Amount of pressure.
   - D. Length of exposure.
   - E. Method and duration of decompression.

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2. Active Treatment.
   (*a*) Recompression.
   (*b*) Palliative and adjuvant measures.

1. *Prophylactic.*

A. Selection of men of suitable age and circulatory efficiency, with the exclusion of the fat, the anæmic, and the arteriosclerotic, are important factors which have been already discussed. Hill says "skinny men are the best for high pressure work."

B. The hygiene of the workers demands attention. Plenty of sleep, a nutritious diet (with but little of fatty food), avoidance of alcoholic and other excesses, exercise of the limbs (during and after decompression) and regularity of the bowels, are important rules for air workers.

The locks during decompression become very cold and either they should be heated or the men should be protected by extra clothing. Hot coffee and warm dressing-rooms and baths should be furnished the men after leaving the compressed air.

C. The amount of pressure is of course the greatest etiological factor, but at the same time it is the one factor which is absolutely beyond control if work is to be accomplished at great depths.

Greenwood[12] safely underwent a pressure of +92 lb. in an experimental chamber, and Haldane reports that a diver descended to pressure of +92.4 lb. without accident, but probably +50 lb. must still be considered practically a maximum pressure for construction work although Hill believes that for short exposures of picked workers, +60 lb. is feasible.

D. Length of exposure means, practically, length of shifts.

The question of the proper limits for working shifts at different pressures is still open to debate, and calls for unbiased consideration, for one must avoid, on the one hand, imposing unfair risk of injury, on the working man, by too long exposures; on the other hand, it is unfair to the contractors to stipulate unnecessarily short working periods.

It is well established that very brief exposures, *e.g.*, up to ½ hour are attended with a minimum of risk; hence lock-tenders, engineers, inspectors, and time-keepers, by reason of their usually brief exposures are much less apt to develop symptoms than are the hard-working men on a regular shift, but of course similar conditions can never obtain in the two classes.

It is interesting to compare different views on this question:

1. Air-workers' union. Stipulations for caisson work.

3¾ and 3¾ hours	to +22 lb.
3 and 3 hours	to +33 lb.
2 and 2 hours	to +35 lb.
1½ and 1½ hours	to +40 lb.
40 and 40 minutes	to +45 lb.

2. Rules proposed by 21 engineers, contractors and doctors and submitted to the N. Y. State Labor Commission.

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5 and 5 hours	to +20 lb.
8 hours with ½ hour for lunch	to +32 lb.
3 and 3 hours	to +40 lb.

3. Ordinary time limits for divers (Admirality Committee).

Over 3 hours	to +26½ lb.
3 hours	to +29½ lb.
1½ hours	to +34½ lb.
1 hour	to +40 lb.

4. Haldane who was on the Admiralty Committee, however, prepares his decompression table for caisson and tunnel work on the basis of two 3-hour shifts up to +45 lb. pressure. We personally believe that two 4-hour shifts up to +32 lb., and 3-hour shifts up to +35 lb., and two 2-hour shifts up to +40 lb. are not unreasonably long, nor unduly short, and we believe that proper decompression will permit longer shifts.

Keays believes that 6 hours continuous shift is preferable to two 3-hour shifts as it exposes the man to the risks of only one decompression instead of two, for he considers that the man is practically saturated in 3 hours and therefore the prolongation of his exposure does not add to the saturation.

E. Method and Duration of Decompression.—This is by far the most important element in prophylaxis, and as it is a matter fully under control we look to it for the best possible solution of the whole question.

The two methods of regulated decompression are known as the Uniform and the Stage methods.

1. *Uniform decompression* is the older and almost universally practised method and consists in reducing the pressure at a constant and uniform rate, *e.g.*, 1 lb. per minute, 2 lb. per minute, etc.

I will mention some practical illustrations; to wit,

	Max. pressure	Uniform decompression
1839, Douchy mines	+37½ lb.	30 minutes
1885, Bridge over Eider	+39 lb.	5½ minutes
1895, Nussdorf bridge	+37½ lb.	35 minutes
*1906, Amsterdam viaduct	+30 lb.	32½ minutes
1908, East River tunnels	+42 lb.	21 minutes (2 lb. per)

In Holland, 1906, legislation was passed as follows:

For +15 lb.	15 minutes
For +22½ lb.	22½ minutes
For +30 lb.	32.5 minutes
For +37½ lb.	42.5 minutes

A few years ago New York State adopted the following regulations for tunnel work:

*The Amsterdam work was conducted on this basis and up to the maximum of +30 lb. pressure; there were no fatalities but there were 229 cases of "bends."

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NEW YORK STATE LAW FOR AIR WORK

Gauge pressure, pounds	Time under pressure	Interval between spells	Uniform decompression, minutes
0–28	8 hours less interval	30 consecutive minutes spent in open air	18⅔
28–35.99	2 spells of 3 hours each	At least 1 hour	24
36–41.99	2 spells of 2 hours each	At least 2 hours	42
42–45.99	2 spells of 1½ hours each	At least 3 hours	46
46–49.99	2 spells of 1 hour each	At least 4 hours	50

New York State Law for Caisson Work, in force Sept. 1, 1912:

TIME OF DECOMPRESSION FOR CAISSONS

Uniform decompression	Time of decompression (in minutes)
Up to 10 lb. pressure	1
Up to 15 lb. pressure	2
Up to 20 lb. pressure	5
Up to 25 lb. pressure	10
Up to 30 lb. pressure	12
Up to 36 lb. pressure	15
Up to 40 lb. pressure	20
Up to 50 lb. pressure	25

The incidence of illness and the mortality has veen very considerable under the older method of decompression (see above).

2. *Stage Decompression.*—Haldane, together with Boycott and Damant, as a result of exhaustive study and experimentation, have quite recently put forward a different method of decompression, the aim of which is still further to minimize the dangers of compressed-air work.

This method, known as the "Stage" method, has been adopted by the British Admiralty Committee on Deep Sea Diving.

A brief theoretical discussion of decompression will best explain the different methods.

Other things being equal, for the same difference in pressure, de-saturation of the tissues (during and after decompression) will occur at the same rate and in the same time that obtained for saturation.

We have already mentioned that saturation does not occur at a uniform rate but is much more rapid at first, *i.e.*, 22 per cent. in 5 minutes, 50 per cent. in 25 minutes, whereas 90 per cent. for the slower tissues requires 4 hours, and for complete saturation at least 5 hours according to Haldane, or 9 hours according to Bornstein.

Therefore if de-saturation takes as long as saturation, it is at once evident that every man who has worked at +28 lb. pressure for 4 hours and then decompresses in 18 minutes (see N. Y. State Law), must emerge from the lock very incompletely de-saturated; in fact Mr. Japp[13] calculates that he will have a maximum air saturation of his body, an emerging, of 25.7 lb.

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Practically, universal experience in compressed-air work has demonstrated that no cases of illness, or a very few minor cases, will develop even after the most rapid decompression from pressures up to +20 lb. Allowing 5 lb. as a margin, Haldane concludes that it is impossible for any bubbles to form after decompression however rapid, following exposures up to +15 lb. In terms of absolute pressure this amounts to a sudden decompression from 30 lb. to 15 lb. or from 2 atmospheres to 1 atmosphere.

Haldane[7] continues, "now, the volume of gas capable of being liberated on decompression to any given pressure, is the same, if the relative diminution or pressure (absolute) is the same."

To give an example: the reduction of pressure from 45 lb. gauge pressure to 15 lb. gauge pressure which is a reduction from 4 atmospheres to 2 atmospheres absolute, will liberate the same volume of gas as the reduction from 15 lb. gauge to atmosphere (or from 2 to 1 atmosphere absolute). If no bubbles can form in the latter instance, none will form in the former; hence Haldane concludes "that the absolute air pressure can always be reduced very rapidly, without risk, to half the absolute pressure at which the tissues are saturated;" but from this point onward the decompression must be very slow, in order that the air pressure in the tissues may never be more than twice the gauge pressure.

Further as Boycott says, this method makes fullest use of the permissible difference of pressure to get rid of the nitrogen from the tissues.

As a result of his studies and his experiments with divers, Haldane offered, and the Admiralty Committee adopted the following "stage decompression" table, based on the assumption that the most rapid decompression from 19 lb. gauge pressure is without risk, *i.e.*, from 2.3 atmospheres to 1 atmosphere absolute.

Dr. Haldane's Rate of Decompression in Caisson and Tunnel Works.

(Extract from report of a committee appointed by the Lords of the Admiralty on deep water diving, August, 1907; Eyre and Spottiswoode.)

Working pressure per square inch, pounds	Number of minutes for each pound of decompression after the first rapid stage		
	After first 3-hour exposure, minutes	After second or third 3-hour exposure, showing an interval for a meal, minutes	After 6 hours or more of continuous exposure, minutes
18–20	2	3	5
21–24	3	5	7
25–29	5	7	8
30–34	6	7	9
35–39	7	8	9
40–45	7	8	9

*In this table, the first rapid stage of decompression, down to one-half the absolute pressure, is to be accomplished in 3 minutes.

Another method suggested by Haldane for tunnel work is illustrated by

*See diagram of Haldane's Method I.

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the following. Where possible, a part of the tunnel is maintained at a pressure equal to half the absolute pressure in the heading. The men now decompress rapidly to this half pressure, remain in this "purgatorial chamber" until the air pressure in the body falls to 19 lb., and then come out by a second 3-minute decompression. (See diagram of Haldane's Method II.)

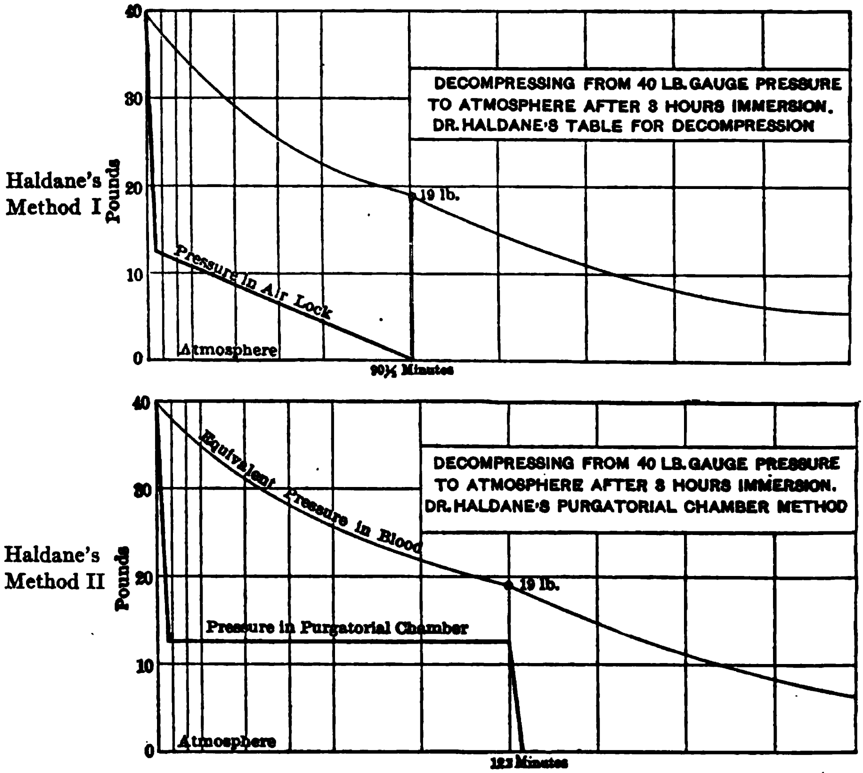

FIG. 5.

In view of his conception of decompression, Haldane condemns "uniform decompression" on three separate counts; viz.: (1) no use is made of the possibility of hastening the exit of nitrogen from the tissues by putting on the greatest permissible stress; (2) "uniform decompression," to be safe, must be so prodigiously slow that it is an irrational proceeding; and however slow it be, the difference between the tissue pressure and the gauge pressure must become larger and larger; (3) following a short exposure, this method merely prolongs the exposure to effective pressure.

The logic of Haldane's "stage decompression" seems irrefutable and Boycott and Damant, experimenting with goats, employed both methods. Their results showed fewer cases of illness (18 per cent.) and no deaths, with the stage method, whereas there were more cases of illness (53 per cent.) including two deaths, with the uniform method. The greatest advantage of the stage method was noted after short exposures.

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On the contrary, Hill and Greenwood, using pigs and rabbits, found no evidence of superiority of the stage method over the uniform decompression.

Hill[12] concludes that there is some evidence in favor of stage decompression after short exposures, but not a decisive superiority after long exposures, and he adds that "the theory is a captivating one, but experiment has not brought that conclusive support which was to be expected."

At the Elbe tunnel the two methods were tried out in caisson work at +30 lb., but very little advantage could be demonstrated for the "stage" method as compared with the "uniform."

Mr. H. Japp,[13] managing engineer of the East River tunnels, recounts the entire freedom from serious and fatal cases in a series of 23,000 decompressions from +40 to 42 lb. at a period in the construction of the tunnels, when a modification of the stage method was enforced by reason of the erection of two extra bulkheads (with air locks) at intervals of 1000 ft.

Three hundred and thirty men were employed for 36 days, working 3 hours on, 3 hours off, and 3 hours on, and were decompressed in the following manner.

Five minutes were spent in the first air lock in decompressing from +40 to +29 lb.; then followed a walk of 1000 ft., occupying 10 minutes before entering the second lock where 8 minutes were spent in reducing the pressure from +29 to +12½ lb. (which latter is half the original absolute pressure). Another 10 minutes was spent in walking to and waiting for the third lock, in which 15 minutes were employed in reducing from +12½ lb. to atmospheric pressure.

Thus a total of 48 minutes was taken for decompression which would have required 90½ minutes by Haldane's method (1), or 123 minutes by Haldane's method (2); and Mr. Japp calculates that the maximum air saturation of the workers upon leaving the last lock was 27 lb.

Hence Mr. Japp concludes that it is practically safe to decompress rapidly from pressures up to +27 lb., and has prepared the following tables for "stage decompression," by which the maximum air saturation of the tissues upon emerging shall never exceed 25 lb.

Decompression Table Based on 25 Lb. Maximum Air Saturation of Body on Emerging
(Prepared by H. W. Japp)

Gauge pressure, pounds	Reduce pressure in 3 minutes to—pounds	Total time in air lock after 8 hours' work, minutes	Total time in air lock after 3 hours' work, minutes	Total time in air lock after 2 hours' work, minutes	Maximum air saturation of body on emerging, pounds
27	6	9	....	....	25
30	7½	24	....	....	25
32	8½	33	25	....	25
35	10	....	35	....	25
40	12½	....	48	....	25
42	13½	....	51	37	25
45	15	....	....	42	25
50	17½	....	....	48	25

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COMPARISON OF UNIFORM AND STAGE DECOMPRESSION

Tunnel pressure (gauge), pounds	Time worked, hours	Uniform decompression (New York State Law) minutes	Maximum air saturation of body on emerging, pounds	Stage compression (based on Table above), minutes	Maximum air saturation of body on emerging, pounds
28.0	3¾ and 3¾	18⅝	25.70	14	25
36.0	3 and 3	24	30.25	36	25
41.99	2 and 2	42	31.25	37	25
45.99	1½ and 1½	46	32.00	35	25
50.0	1 and 1	50	32.50	33	25

Japp's table provides a great shortening of decompression as compared with Haldane's tables, which latter, Hill also agrees are unnecessarily long. As compared with the New York State uniform decompression table, there is also a slight shortening in time, but a marked advantage in the maximum air saturation of the tissues, on emerging.

Hill recommends one stage at +8 lb., lasting 15 minutes, after a shift at +30 lb., and 5 minutes given to completing decompression; also one stage of 30 minutes at +15 lb. after a shift at +40 to 45 lb., with 10 minutes spent in completing decompression.

Von Schrötter[6] recommends from +30 lb. to reduce in 3 minutes to +12 lb. and then takes 32 minutes to complete; or from +45 lb. reduce to +22½ lb. in 3 minutes and takes 60 minutes to complete.

Conclusions.—No one of the suggested methods of decompression is efficient in entirely eliminating cases of "bends," but the improved methods should certainly minimize the serious and fatal cases.

Indeed in Mr. Japp's series of 23,000 decompressions by the modified stage method, there was an unusually large percentage of minor cases; for, reporting on 8510 of these decompressions, Keays found 1.6 + per cent. of minor cases, and only seasoned men were employed.

However the writer believes that Mr. Japp's table is reasonably safe and is the most practical of those yet devised.

Exercise during and after decompression, also the inhalation of oxygen in the last period of decompression and following it, are claimed by Hill and others to greatly hasten the elimination of nitrogen. (N. B.—The inhalation of pure oxygen under pressures above +30 lb. is irritant to the lungs and highly dangerous.)

2. *Active Treatment.*

(*a*) *Recompression.*—Had infectious diseases as wonderfully a specific treatment as has compressed-air illness, the practice of medicine would be revolutionized.

To behold a man paralyzed from the waist down, carried into the medical lock, or recompression chamber; the pressure raised, and within 5 minutes to see this man get down from his couch and walk about, is indeed little short of miraculous and yet this is of no uncommon occurrence.

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Recompression as a method of treatment arose, first, among the workers themselves, who found by experience that their pains were relieved upon re-entering the caisson or tunnel.

To-day every properly equipped compressed-air works should be supplied with one or more specially constructed recompression chambers called "medical air locks," which should be arranged with two compartments, thus permitting the medical attendant to enter or leave without disturbing the pressure in the medical chamber. Such locks are fitted with electric lights, steam heat, clock, pressure gauge, telephone, and comfortable couches, also there is a glass port in the door which allows observation from the outside.

1. Theory of recompression.

If symptoms are due to the liberation of bubbles of nitrogen in the tissues and body fluids, then recompression will at once reduce the size of these bubbles and moreover, will urge them again into harmless solution in the tissues, and if decompression now be carried on at an appropriate rate, the nitrogen will be given off into the alveoli, by means of the circulating blood and without the formation of bubbles.

2. Degree of pressure for recompression treatment.

At the East River tunnels, it was the practice to raise the pressure to the equivalent of the tunnel pressure, and then at once to start decompression at a rate not exceeding 2 minutes per pound; but the best results were obtained by a rather rapid decompression from *e.g.*, +32 lb. to +15 lb. and thereafter very slow reduction to the atmospheric pressure, 60 to 90 minutes or even two hours being sometimes thus employed (This will be seen to be a modification of the stage decompression).

Other men (L. M. Ryan[17]), claim that good results are obtained by raising the pressure to only two-thirds of the working pressure, and allowing 1 hour for reduction from +15 lb., and 3 to 4 hours for reduction from +20 lb., in severe cases.

In criticism of Ryan's suggestion, although admitting that pains will often disappear long before the full pressure is reached, the writer believes with Haldane that the first rapid stage of decompression from the full pressure will aid "the exit of the nitrogen by making use of the greatest permissible stress;" and therefore full pressure should be employed.

3. Time of administration of recompression treatment.

By far the best results attend recompression when instituted as soon as possible after the onset of symptoms, although partial or complete relief often is obtained after a lapse of hours or even a whole day.

4. Practical results of recompression.

Keays[8] showed that, in our 3692 cases, about 90 per cent. were completely relieved by recompression. Some required two or more treatments, but 68 per cent. were relieved by a single recompression.

About 9 per cent. more were partially relieved and only 0.5 per cent. failed to get any relief.

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Even in the cases of partial paralysis of the legs (18 cases) 12 cleared up entirely with one recompression; and of the 16 cases of complete paraplegia, seven were cured by one recompression.

Of cases with collapse and unconsciousness (17 cases), eight were relieved by one or two recompressions, but nine were fatal.

(*b*) *Adjuvant and Palliative Measures.*

Exercise and massage and the inhalation of oxygen both in and out of the medical locks will aid elimination of nitrogen.

Counter-irritation, heat, electricity, liniments and massage may suffice in many minor cases of "bends "even without recompression, or may be used as adjuvants in refractory cases.

Ergot was recommended by Dr. A. H. Smith,[5] but I have never seen any benefit from its use, and moreover the indications for its administrations were based upon the discarded mechanical pressure theory.

Symptomatic medical treatment will be indicated; *e.g.*, anodynes in cases of unrelieved pain and hypodermatic stimulation in cases of collapse with impending circulatory or respiratory failure.

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CHAPTER IV

EFFECTS OF DIMINISHED ATMOSPHERE UPON HEALTH

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The effects of diminished atmospheric pressure and physical exertion in mountain climbing upon the heart and respiratory organs, characterized by rapid and difficult breathing, palpitation of the heart, headache, dizziness, faintness, nausea, extreme muscular fatigue, and occasional hemorrhage from the nose, gums and ears, have been known for some time. These symptoms have been observed in persons not accustomed to a rarified atmosphere in altitudes of 2000 to 3000 meters, and higher elevations naturally intensify the effects.

While some of these effects may be clearly traced to diminished atmospheric pressure and deficiency of oxygen, muscular exertion and fatigue toxins doubtless also play an important rôle. Mountain climbing involves lifting the entire weight of the body through the distance climbed, which for a man weighing 175 lb. is equivalent to about 8 ft.-tons of work for every 90 ft. elevation. Hence work of this character, or any muscular exertion such as mining and construction work, involves increased production of waste material in the blood, and doubtless accounts for the insomnia, loss of appetite and chronic fatigue observed among the miners and other workers in high altitudes. The heart and lungs gradually adapt themselves to the diminished atmospheric pressure, the vital capacity of the lungs is increased in consequence of deeper inspirations, and there is much reason for assuming that the functions of the hemopoietic organs are also gradually increased in high altitudes. The most recent investigations[1] show that the number of red blood corpuscles, the amount of hemoglobin or coloring matter and the amount of oyxgen in the blood are distinctly higher than the "normals" of sea level.[2] In spite of this beautiful adaptation it is evident that the efficiency of workers is greatly impaired; even the native copper miners at Cerro de Pasca, altitude 16,000 ft., according to Oliver,[3] can only work in 8-hour shifts for a few months at a time, when they are obliged to cease and return to their farms.

Ballooning.—It has been generally assumed that in the absence of physical exertion, modifications in the action of the heart and lungs are less liable to ensue in balloon ascensions. Petard in 1873, with four companions, found this to be true, since in a balloon flight the phenomena of so-called mountain sickness were not observed until an elevation of between 4000 and 4690 meters was reached, when the pulse rate was 114 and respiration 44 per minute. Von Schrötter,[4] since 1896, has made a number of balloon

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ascensions and determined that serious symptoms are rarely observed in aeronauts, except in specially predisposed subjects, until an elevation of 5000 meters is reached.

This altitude, and higher elevations, he regards as the danger zone since, in addition to the cardiac and respiratory symptoms, there is also great mental depression, and the slightest muscular effort, such as unloading a sack of ballast, is followed by faintness. In elevations above 5000 meters the pulse, at first rapid, becomes small and irregular. Dr. Flemming and Steyrer,[4] in June, 1911, attained an elevation of 8910 meters and experienced extremely grave symptoms, such as diminished cardiac and respiratory action, pulse almost imperceptible, cramps and tremblings of the muscles, diminished mental and spinal sensibility, cyanotic countenance, and unconsciousness—symptoms urgently calling for the use of oxygen inhalations. Most authors agree with Zuntz that, while these symptoms are caused by a deficiency of oxygen in the system, the diminished atmospheric pressure also produces a gaseous distention of the intestines which impinge upon the diaphragm and seriously interfere with the vital capacity of the lungs. Nausea with occasional vomiting, in fact all of the symptoms of seasickness, is liable to occur at any elevation if the movement of the airship, in consequence of irregularities of the winds and eddies, has become unsteady.

According to Dr. Reymond,[5] the effects of aviation on the ear are characterized by a painful tension, ringing or buzzing noises and impaired hearing. These phenomena are due to differences in atmospheric pressure, and hence are more pronounced during sudden changes in elevation and after a rapid descent. The noise from motors tends to aggravate the effects. Airmen wear suitable caps with ear mufflers, and some have also resorted to plugs of cotton, but this additional safeguard had to be abandoned as it interfered with observing modifications in the action of the motor. Ordinarily the act of yawning or swallowing, with mouth and nose closed, serves to restore the equilibrium between the inner and outer air of the internal ear. The temporary deafness varies in different individuals and would appear to go off with practice, but Reymond found that cases of catarrh of the Eustachian tube are greatly aggravated, especially in mouth breathers, on account of the strong winds, and require more active treatment.

The effects of strong air currents and intensive insolation upon the eyes are harmful. The activity of the violet and ultra-violet rays is greatly increased in high elevations, but there is no evidence that the sense of vision is in any way impaired, if protective glasses are worn.

Sunburns and severe forms of dermatitis caused by intensive insolation are more common in high elevations, but may be prevented by suitable hoods and protection of the hands and arms. The excessive dryness of the upper strata of the air causes rapid evaporation of perspiration and pulmonary exhalation, and may produce inflammatory conditions of the skin and mucous membranes, especially of the eyes and upper air passages.

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The effects of sudden changes in temperature and exposure to extreme cold, rain or hail upon the system cannot fail to prove injurious. Von Schrötter reports flights in which the thermometer registered $-40°C$. Fortunately the air is practically frozen dry and the effects of cold are less intense. Nevertheless exposure to extreme cold, fogs and atmospheric electricity, and the modifications in respiration, etc., are doubtless frequent causes of accidents.

Among the occupational risks of ballooning should be mentioned the possibility of "gas poisoning." Von Schrötter[6] refers to several instances of carbon monoxide poisoning which occurred during the inflation, deflation and collisions of balloons. Prof. Glaister[7] of the University of Glasgow reports 16 cases of poisoning from arseniuretted hydrogen gas which occurred, in ballooning for military and other purposes, during the inflation or deflation of the balloons or from leakage during balloon flights (see page 8). Similar cases have been described by Dr. Maljean[8] of the French Army and Crone[9] of the Prussian Army.

Dirigible Balloons.—Since the introduction of motors in dirigible balloons, new elements of danger have been added, such as gasoline explosions, breaks in the propeller, failure to get gasoline into the cylinder during a steep incline of the apparatus, etc. But, after all, the accident liability in balloon flights is not as great as has been generally assumed. According to Flemming, cited by von Schrötter, during the year 1908 there were 1713 balloon flights in Germany in which 5786 persons participated; of these, 50 persons were injured, four or 0.07 per cent. resulted in death, 24 or 0.41 per cent. in serious injuries, and 22 in slight injuries.

Aviators.—The percentage of fatal injuries is very much greater among aviators. We have no precise data, but the statistics compiled up to April 1, 1914, show that 462 aviators have been killed since September 17, 1908, when the first man, Lieut. Selfridge, U. S. A.,[10] lost his life.

According to Dr. Reymond[11] "the greater number of accidents of aviation are due to the apparatus, to the imprudence or the clumsiness of the aviator, to his physiological condition, which may itself be caused by the conduct of the apparatus; but it would be premature to draw conclusions as yet." The physiological effects of diminished atmospheric pressure upon the lungs, heart, etc., probably because of the more rapid flights, are observed in very much lower elevations than in balloon ascensions. Some interesting observations were made on this point by Cruchet and Moulinier[12] during the aviation contest at Bordeau in September, 1910. They found at an elevation of 1000 meters an increase in the blood pressure of between 30 and 40 per cent., and the action of the heart was quite rapid. These symptoms were not noted in elevations between 100 and 150 meters. None of the flights exceeded an elevation of 3000 meters, and symptoms such as palpitation of the heart, earache and ringing in the ears and vertigo were especially marked in rapid descents. Flemming[14] reports two cases of unconsciousness

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in aviators, which occurred at an elevation of between 100 and 200 meters and which he attributed to the combined influence of cold, flatulent distention of the bowels and passive congestion, caused by tight-fitting clothing.

My friend Mr. Douglas McCurdy, one of the most expert and intelligent of American aviators, in commenting upon the effects of exposure to extreme cold writes me as follows: "I had an experience while flying at Baddeck, Nova Scotia, during the month of February, 1909. The temperature was about zero and of course I was warmly clad. I had flown about 10 miles, landing at my starting point, when my hands seemed unusually cold. I then felt a most curious feeling come over me, where people and sounds right around me seemed distant. I broke into a violent perspiration and was helped into a doctor's sleigh, which was near by, and covered up with warm robes. This most delightful sensation lasted for about 5 minutes when I gradually became normal again."

The psychic factors in aviation have recently been studied by Loewy and Placzek,[13] who report a series of tests of the attention and other psychic phenomena on themselves and two others in a pneumatic cabinet with an atmospheric pressure corresponding to that of an altitude of 4000 meters. The findings are summarized as follows: The objective findings were comparatively slight, although mistakes in doing sums were more frequent the more rarified the air. But the sensation of being incapable of giving close attention, of being unable to act promptly and with precision—these subjective factors were pronounced, and even a subjective sensation of the kind has always more or less of a paralyzing effect and may serve to explain some of the fatalities to aviators for which the machines were not responsible. The effects in these tests were the more significant as the subjects were quiet, warm and free from responsibility in the pneumatic cabinet. "Compare this with the condition of the aviator," they remark, "the rapid changes in altitude causing physical disturbances and weakening the will power, while the wind makes it hard to breathe and chills the surface, and finally paralyzes the peripheral vessels so that the blood pours into them and the brain, etc., become anæmic." They think that the death of Chavez, for instance, was due to these causes, as he strove to break the altitude record. They warn aviators against high altitudes and urge them to train themselves to bear abrupt changes in atmospheric pressure.

Mr. Earle L. Ovington, an expert high flyer in the United States, published a valuable criticism of these researches which may be summarized as follows: He has repeatedly flown at heights in excess of 4000 meters, (about 2½ miles), believes in high flying and considers it less dangerous than low flying, because the vertical air currents which are dangerous at low altitudes have no effect at great heights. He believes there is a great difference in the psychologic condition between two men cooped up in a little cabinet under artificial conditions and the same men flying free through the

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clear atmosphere at a height of 2 miles. He does not believe that the rarified air paralyzes the action of the subjective mind, and his experience has been that the objective mind, at least, is stimulated by the excitement of rushing through the pure air at high speed, and possibly by the greater actinic power of the sunshine in high altitudes. Mr. Ovington has had no difficulty in breathing in an aeroplane at a speed of over 150 miles an hour (with the wind, of course), and states "that an aeroplane properly designed is so constructed that a great deal of this wind is done away with." He has not found that rarification of the air interferes with breathing owing to the fact, as he believes, that the aviator climbs so slowly that he has plenty of time to get used to the diminished atmospheric pressure. He describes the bad effects of rapid descents as follows: "If I cut off my engine at a 2-mile height and drop suddenly to a mile from the ground, it seems as if some one was standing behind me poking red hot pokers into my ears. . . . My usual practice, when I wished to avoid this inconvenience, was to drop 1000 ft. as slowly as possible with the power off, turn on the power and circle around at that level, swallowing vigorously. I would then drop another 1000 ft. In this way I could get to the ground from a height of 2 miles without any inconvenience. . . . At Columbus I thought I would do a little grand-stand work and drop from the height of a mile straight to the ground as rapidly as possible. I stepped out of the machine with everything swimming before my eyes and just as my foot touched the ground I became unconscious and remained unconscious for 5 minutes or so. This was no doubt due to the rapidity of my descent, and this may have caused Chavez to lose control of his machine when only 100 ft. from the ground."

Whatever difference of opinion there may be as to the ability of the mind and body to accommodate itself to the effects of diminished atmospheric pressure, and much of this doubtless depends upon inherent qualities of the aviator, prudence demands that rapid ascents and descents should be avoided.

That there is special danger in sudden descents is apparent from the facts already stated, and is also evident from the following personal note by Mr. McCurdy upon the death of Archie Hoxey at Los Angeles, California. He writes: "Mr. Hoxey was one of the most expert of flyers, and had given a good deal of attention to high flying (about 11,000 ft.). He had experienced at several times a dizzy sensation and a slight feeling of nausea upon landing, due we thought to a too rapid descent. We cautioned him and advised a slower rate of descent, but he paid no heed. At the time of which I speak he had attained an altitude of about 11,000 ft. and came down very rapidly to about 300 ft., when the machine was observed to continue an abnormal descent and Mr. Hoxey seemed to have abandoned his controlling levers. The machine finally struck the ground and Hoxey was killed. He had made apparently no effort to land normally."

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Preventive Measures.—The general symptoms observed in ballooning and aviation clearly indicate that airmen should have sound eyes, ears, heart and lungs. Dr. Reymond refers to an aviator with a heart lesion, and another afflicted with pulmonary tuberculosis in the second stage; in both of these cases the symptoms were not aggravated by the continuous practice of aviation; "asthmatics and emphysematous subjects, on the other hand, suffer very much from the wind raised by the screw or from the speed of the relative air current."

Referring to the effects of aviation on diseased lungs, Mr. McCurdy informs me that Mr. Hubert Latham of France was practically given up by his physicians as he was suffering from consumption. He therefore decided that he might as well have some excitement for the remaining few months of his life by flying. He became connected with the Antwanette Co., the makers of the beautiful monoplane which bears their name, and soon became the most expert flyer in the world. He was in the open air so much that he almost completely recovered his normal health and lived for several years. He was, however, killed by a wild bull while hunting in Madagascar in 1912.

It is apparent, however, that only physically sound persons should engage in this work, which is full of possibilities in the future. All ascending and descending flights should be made gradually and all elevations above 1500 meters should be avoided. A reserve pilot should always be on duty to render first aid. An apparatus for the administration of oxygen is indispensable. Dr. E. Koschel[15] reports that his pulse in the flights of 1911 was 140 at an elevation of 5000 meters and 160 at an altitude of 6000 meters. In 1912 by resorting to timely oxygen inhalation, at an elevation of 3500 meters, he attained and remained in altitudes between 7500 and 9000 meters, with a pulse not exceeding 100 per minute and was perfectly capable of physical exertion, such as unloading sacks of sand ballast. Protection for the head and eyes is necessary not only against excessive insolation but also in case of rain, hail and snowstorms.

Flemming has pointed out the dangers of tight-fitting clothing. Several thicknesses of light woolen clothing, loosely draped with some impermeable outer garment, woolen socks and felt boots offer the best protection against cold and winds.

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DIVISION II

Systemic Occupational Diseases

CHAPTER I

DUST DISEASES OF THE LUNGS

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That pulmonary disease develops with greater frequency in persons following certain occupations compared with others has long been known. The circumstance did not escape the attention of Ramazzini 200 years ago. A century ago physicians in Great Britain were more familiar with the phthisis of coal miners than is the present generation. Colliers became the subjects of cough, they emaciated, they expectorated large quantities of an inky black material, and after death the lungs were found to be absolutely black and cavitated. Our knowledge of the disease goes back to 1815 when Pearson presented a paper to the Royal Society of London upon the subject. In 1831 Gregory and Christesin of Edinburgh extracted from the lungs of coal miners dying from phthisis black material which they found to be carbon. The dark color of miners' lungs had been regarded by Laennec as the result of smoke from the lamps, but by Behier in 1837 and Rilliet in 1838 it was attributed to particles of carbon. Belgian colliery doctors, among whom Gobert of Hainaut (1827) and Kuborn of Liege (1861) may be mentioned, were familiar with the asthma of miners and its accompanying black spit. Another explanation of the discolored lungs of miners was offered by Virchow. The pigmentation arose according to him from a transformation of the hematine of extravasated blood. It is needless to say that Virchow's theory has few exponents.

The malady is known as *anthracosis* or *coal miners' phthisis*. Miners working in certain coal fields are more affected than others, a circumstance which suggests that the physical character of the coal plays an important part in the development of the illness, some coals being harder than others. From a historical point of view the opening out of the South Wales coal fields is a modern development compared with those of Northumberland, but owing to the smokeless character of the coal and its suitability for steamship purposes the industry received an impetus half a century ago which has not yet begun to decline. When Sir John Simon wrote upon the subject nearly 50 years ago he was of the opinion that there was a larger amount of bronchial and respiratory disease among the Welsh miners than among the Northumber-

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land, a circumstance which he attributed to the better ventilation of the mines in the North of England. To-day ventilation of coal mines in all the civilized world is good and as a consequence the health of coal miners is satisfactory.

This is particularly so as regards tuberculosis. British coal miners do not exhibit a high mortality rate from this disease compared with men working in most other trades. Taking the tuberculosis mortality figures of all occupied males in England and Wales as 175, that of coal miners is only 85. In the United States the death rate from all causes among miners and quarrymen (the two occupations are linked together) is 120.9 per 100,000 living, for lawyers 139.9, for physicians and surgeons 168.8, for carpenters and joiners 231.0, and for marble and stone cutters 540.5. According to the same census, pulmonary consumption caused 10.6 per cent. of deaths of all miners and quarrymen as against 16.2 for all occupied males. At Scranton,[1] U. S. A., during 10 years pulmonary tuberculosis caused 3.37 per cent. of all deaths and among all other occupied males 9.97 per cent. Of lung diseases alone tuberculosis caused 14 per cent. of all deaths among anthracite miners in Scranton and 41 per cent. of deaths among other occupied males. This comparative freedom of the coal miner from tuberculosis has raised the question as to whether some protective influence is not exercised by coal dust upon the lungs. From their experiments upon animals Wainwright and Nichols were disposed to attribute a certain amount of protection to the lungs against tuberculosis. Nichols regards the calcium salts in coal dust as the protecting agent. It is certainly astonishing, for example, how rapidly wounds in coal miners heal.

While we admit that the comparatively good health and longevity of the coal miner are the result of the improved ventilation of recent years, there is just the possibility that excessive ventilation by overdrying the air of mines raises and carries onward very fine dust which may not only do damage to the lungs of those who inhale it but may also become a cause of explosion in the mine. If the work of a coal miner is hard, his hours are not long compared with other occupations and the atmosphere in which he works, if in the line of the intake air, is satisfactory. It is when men are working at the face, in confined places and away from the immediate delivery of air, that they are liable to be injured by dust. The more confined the space and the less readily the atmosphere can be cleared of fume and dust raised by the use of explosives to bring down the rock, the greater is the liability of the men to become the subjects of pulmonary disease. To the various forms of disease of the lungs caused by inhalation of dust the term *pneumokoniosis* was applied by Zenker—from the Greek word *pneumon*, lung, and *konis*, dust. Although the kind of dust inhaled directs the type of the pulomonary lesion, yet one pathological substratum underlies all forms of the disease, viz., fibrosis or a replacement of the elastic tissue of the lung by an unyielding fibro-connective tissue. The fibrosis imparts solidity to the lung and abridges

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its aerating function. To the black lung of the coal miner as already stated the term *anthracosis* is applied, to the gray-black lung of the stone cutter *chalicosis* is given, to that of the gold miner *silicosis*, and to the lung of the worker in metal dust the term *siderosis* is applied. The lungs of persons who live in large towns are always more deeply pigmented than those of persons living in the country, and on microscopical examination the cause of the pigmentation is invariably found to be particles of carbon or other kinds of dust. In a dusty atmosphere it is the finer particles which reach the lungs and damage them. The heavier particles are apt to be caught in the nares and upper portion of the respiratory tract and to be expelled in coughing. The ciliated epithelium of trachea and bronchii is a defensive barrier to the lungs, since dust caught in the mucus secreted by the tubes is by the waving action of the ciliated epithelium wafted outward. Recurrent colds lead to a shedding of the ciliated epithelium and with this desquamation an important defense is lost to the lungs. This circumstance is not without importance since recurrent catarrh of the upper respiratory passages is a frequent prelude to pneumokoniosis.

So far as coal miners' phthisis is concerned it may be stated that a collier who develops anthracosis may live for years and be little inconvenienced by his malady. This applies only to the man who has worked in soft coal. It is otherwise if the coal has been of a hard, gritty and stone-like nature. Miners keep bringing up large quantities of black spit for years after they have ceased working in coal—a circumstance which shows not only that considerable deposits of carbon had taken place in the lungs, but that the lungs tend to rid themselves of the deposited carbon largely by means of the action of pulmonary phagocytes. It cannot be said of coal miners that they are an unhealthy class of men. Far from it; apart from the risks of mining their prospects of longevity are good. In a country like South Africa a comparison can be made between the harmlessness of coal dust and the power for harm of the rock dust of the gold mines. Dr. F. I. Allen, who has been for several years Medical Officer to the leading Transvaal collieries, states that although anthracosis is common among coal miners, it does not impair the health nor the working capacity of the men compared with the silicosis of the gold miner. This circumstance confirms what has already been stated, that the physical character of the dust is an important factor in pneumokoniosis. The fibrosis present in the lungs of persons who have worked in a dust atmosphere of itself suggests that we raise the question whether the fibrosis is the consequence of irritation induced by foreign particles inhaled, or whether the foreign particles seen in the lungs after death have not been caught and retained in the lungs owing to those organs having undergone a fibrotic change through the operation of some other cause than dust. For a comprehensive study of the relation of pulmonary fibrosis and dust Dr. Shufflebotham's Milroy Lectures will repay perusal.[2] This subject is not less interesting than is that of pneumokoniosis and such microbic

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infection as tubercle to which we refer later. Dust of animal origin as wool, hair, and feathers induces catarrh of the respiratory passages rather than structural changes in the lungs.

In pneumokoniosis so completely replaced is the sponge-like structure of the lungs by solid tissue that it is difficult to understand how respiration is carried on even as well as it seems to be. In some occupations more than one kind of dust is present in the atmosphere. This is the case in the Sheffield steel grinders in whose lungs there may be particles of steel and also particles of stone from the grindstone. Masons who chisel fine sandstone are more prone to phthisis than men who chip and chisel granite where the particles of dust are heavier. It is owing to the larger and heavier size of the particles of dust that the comparative freedom of the Aberdeen granite worker from phthisis is attributed. A change apparently is creeping over this industry. Since the introduction of pneumatic tools for cutting and smoothing the stone greater quantities of fine dust are raised, and as the work is carried on no longer in open sheds there has been recently more lung trouble among the granite workers. The lungs of men who work in oxide of iron are brownish red in color. The structural changes in the lungs in pneumokoniosis commence in the interstitial tissue; subsequently the pleura tends to become involved. Emphysema is also present and there are usually signs of a widespread bronchitis.

The best types of pneumokoniosis are found in persons working in such dusty atmospheres as those of the Sheffield trades, ganister crushing, buhrstone masonry, and the gold mines of South Africa and elsewhere.

Since, practically speaking, the same morbid changes are present in all forms of pneumokoniosis no matter what the particular kind of dust which has caused the disease, it is unnecessary to describe in detail the various forms of pneumokoniosis. Although there is little difference in the pathological appearances presented by a lead miner's lung, a gold miner's lung and that of a Sheffield or other steel grinder, there is setting aside individual idiosyncrasy, not perfect uniformity since in some forms of pneumokoniosis there is during life a greater tendency to pulmonary hemorrhage than others.

Persons employed in dusty trades have a shorter life by a few years than those employed in occupations free from dust. It must not be forgotten that to this shortening of life other circumstances than dust contribute. Buhrstone is one of the hardest stones known. It is used for making millstones for the grinding of cereals, coprolites, etc. It is not a large industry. I have visited the buhrstone yards on the Thames and at Fierte-sous-Jouarre in France. Few buhrstone workmen live beyond the age of 35–40. Most of them are intemperate in the use of alcohol. The men complain that, in the operation of chiselling, the dust gets into the back of the throat and creates an exasperating sense of dryness which stimulants remove. At Fierte-sous-Jouarre I found men, comparatively young, who consumed a bottle of brandy daily in addition to a liter or more of red wine. The men

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who stood the work best and who exceeded 50 years of age were abstainers. Alcohol made the men careless.

Ganister disease is a form of silicosis due to inhalation of the dust of ganister, an exceedingly compact, close-grained and highly siliceous rock. It contains 99 per cent. of pure silica. Owing to the almost adamantine hardness of ganister the rock cannot be won by the ordinary methods of mining. It has to be blasted. The broken rock is placed in powerful grinding mills to be pulverized preparatory to being made into bricks which, since they are capable of resisting extremely high temperatures, are used for lining the interior of iron blast furnaces. The men who mine the ganister, those who grind it and those who make it into bricks all suffer, but in varying proportions, from pneumokoniosis. For every 1000 men employed Dr. Birmingham[3] found that each year

42.3 ganister miners die	or 34.4 per cent.
179.8 ganister grinders die	
22.2 ganister brickmakers die	

179.8 deaths per 1000 ganister grinders is an appallingly high mortality rate. The invasion of the lungs by the dust is generally indicated by signs of bronchitis. As the disease advances the lung becomes the seat of well-marked fibrosis. Once symptoms reveal themselves the disease is found fully developed within 1 year. The miner becomes anæmic and enfeebled and his breathing quickened. There are dyspnœa on the slightest exertion and hacking cough. Few of the patients survive 2 or 3 years. Death is generally due to tuberculosis.

According to Dr. Robertshaw, Medical Officer of Health, Stockbridge, the mortality of ganister miners from pulmonary tuberculosis has fallen 15 per cent. in recent years, while the median age at death has risen from 37 to 45 years, owing to the compulsory use of steam jets in the grinding mills.

According to Dr. Edgar L. Collis, English stone masons have a mortality rate three times greater than that of all occupied males; also while the average age at death used to be 37–38 it has in recent years risen to 43–44. Sandstone is more dangerous than limestone. The mortality rate of stone masons is not the same for every district. This Collis shows is largely due to the amount of silica present in different kinds of stone. The dangerous ingredient is silica. The pulmonary tuberculosis of stone masons comes on at a later period of life than in males generally, and is only slightly infective. A similar absence of the infectiveness of tuberculosis in the Grinskill quarrymen is alluded to by Dr. Wheatley,[4] Medical Officer of Health for Shropshire. He says that there is no excess of phthisis in the wives of men who are victims of the disease.

In the gold mines of the Rand the men who work rock drills driven by compressed air are short lived. Five to 6 years of work bowl most of the

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men over. Silicosis in rock drillers seldom develops until after 3 years' work, but once the disease originates, if the men continue to follow their employment, within other 2 years many of them die. The men who use the hand drill are less liable to be overcome by the dust than those who use drills driven by compressed air. The work is carried on at great depths. Several of the miners go down 3000 ft. and more. The work is fatiguing, the ventilation is bad, the temperature high and the noise is great. After a day's work in the mine the men when perspiring are rapidly transported by means of lifts to the surface where the temperature is several degrees colder than that in the mine. As bronchial catarrh thus induced is given little heed to by the miners, the way is prepared for any dust which is inhaled more readily to reach the lungs. In one sense the men themselves are partly responsible for the pulmonary disease. After firing explosives in the mine to shatter the rock they return too early to the place of work before the fumes have cleared away and the dust has had time to settle. The owners of the mines could do more than they have done to improve the ventilation of the South African gold mines. In some of the mines there is a total disregard of all hygienic measures. Unless greater attention is given to details of hygiene personal and general, medical examinations of the men before undertaking the work avails but little.

Prevalence of Miners' Phthisis in South Africa

In a lecture Dr. Watkins-Pitchford[6] stated that in the opinion of the Transvaal Miners' Phthisis Commission of 1903 probably 21 per cent. of all underground miners were affected with silicosis. In 1912 the Second Commission after examining 3136 underground miners found 990 or 32 per cent. suffering from miners' phthisis in one or other of its stages, and of these about two-thirds of the cases were in the stage of early fibrosis. As the use of the rock drill is regarded as the main cause of the lung trouble it is to be noted that while in December, 1905, there were 1800 of these machines at work, in December, 1912, there were 5600.

Since the gold mines of South Africa supply the best illustrations of pneumokoniosis and the largest number of victims of the malady, and since it is from the men who have worked in these mines and have returned in ill health to Great Britain that my own experience has been drawn, I may be pardoned if a further slight digression is made into the mining and other conditions which prevail in the Transvaal. Both white men and Kaffirs, called "boys," are employed. On the Witwatersrand the average number of white miners working underground in 1912 was 11,607; in this year 161 white miners died from silicosis. Notwithstanding the fact that many invalided miners came home to the North of England, Cornwall and elsewhere the deaths from silicosis in the Transvaal were 13.8 per 1000 for the year.

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The number of natives working underground for 1912 was 149,782 out of an available supply of 192,522; of these men 153 died from silicosis—a small number compared with the white men, but possibly explained by the fact that the natives do not remain long at work but return to their homes.

When we consider that on an average there are 11,840 white miners working underground in the Rand and that at least 32 per cent. of these are affected with miners' phthisis it is safe to conclude that 3700 of these men are the victims of silicosis.

Causation of Pneumokoniosis in General

Inhalation of dust, hard and gritty, also sharply angular is the main cause. In the case of the coal miner it is not the carbon particles of the coal but the stony particles to which the carbon particles are affixed which injure the lung. The pneumokoniosis of the steel grinder is caused by inhalation of particles of steel and sandstone—mostly the latter; the lung disease of the Transvaal gold miner is the result of inhalation of quartzite, a siliceous sandstone which has become hardened and solidified by metamorphosis. Interbedded in the quartzite are shallow beds of conglomerate or "banket" composed of quartz pebbles and pellets of pyrites cemented into a solid mass by silica. The gold is found in a finely divided state in the material which binds the pebbles together. The main object of the gold-producing industry of the Rand is to mine the conglomerate, bring it to the surface, crush it to powder and extract the gold by the amalgamative and cyanide of mercury process. "Silicosis is caused by the inhaling of the dust which arises when the conglomerate as well as the various rocks which have been mentioned are drilled, blasted, shovelled and crushed." To secure the gold-bearing conglomerate the quartzite has to be broken. This is accomplished by drilling holes 4 to 8 ft. deep into the quartzite and placing in the bottom of the holes dynamite cartridges fitted with detonator and fuse. As hand drilling is a slow process the work is now almost entirely done by machine drills, of a percussive and not of a rotary type, driven by compressed air. In an 8-hours shift a rock-driller will bore through 22 to 50 ft. of solid rock, *i.e.*, he will bore from 4 to 8 holes. In order to lay the dust generated during the process of boring the interior of the hole and the immediate surroundings are kept wet with water by spraying. By this means the dust which would otherwise be dangerous is converted into mud. Upwardly inclined holes are with difficulty kept wet. The rock drills used are principally the Ingersoll and the Leyner. It is advantageous to use a rock drill which automatically washes out the hole with water at the same time that it bores. The amount of mud thus formed is considerable. When dried this would again become a source of danger to the men. To obviate this the mines have to be kept as damp as possible. The moisture of the mine and the high temperature in which the men are working are not with-

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out influence in causing fatigue and in reducing the vital resistance of the workers.

During blasting of the rock considerable quantities of fine dry dust are dissipated through the mine. Shovelling of the detached rock and its transference by wagon also contribute to the dustiness of the atmosphere. The fine particles of dust remain suspended in the air a long time; they are capable of passing through very fine sieves so that after the use of explosives it is absolutely essential that the miners should not return too early to that particular part of the mine in which they had been working. According to Mining Regulations half an hour's delay is considered to be long enough if the air is free from dust, smoke and fume as perceived by sight and smell, for miners to return to the working. Thirty minutes is hardly long enough for the fine dust to settle. Somehow or other fine dust seems to take longer to settle in a mine than at the surface. Hitherto gold miners, as they are paid according to the number of feet driven by drilling or sunk in development faces, have in their haste to make large wages disregarded many of the simple rules which make for health.

Symptomatology.—In nearly all the South African gold miners whom I have treated for pulmonary disease the malady has been in existence for several months. Once it is developed the disease may last 2 years or more. There are cases on record of "acute pulmonary silicosis" where after a sharp attack of broncho-pneumonia the patient who had worked only a few months dies. After death the lungs are found to be congested, œdematous and irregularly pigmented without the naked-eye appearance of fibrosis. On microscopical examination of sections of the lungs by polarized light, in the connective tissue are seen myriads of siliceous particles. The pulmonary alveoli are filled with catarrhal cells and serous exudate. It is unusual for miners to die so early. The silicosis for which miners seek advice develops slowly, so insidiously in fact that it is difficult for the patient to assign a date to the onset of symptoms of which dry cough in the morning or on leaving the mine at the end of the day is one of the earliest symptoms. What appears to be a bronchial cold, with or without pleuritic pains, attended by a sense of increasing debility is an indication that the lung has become affected by mine dust. One feature stands out prominently throughout the whole course of gold miners' phthisis, viz., dyspnœa on the slightest exertion—a shortness of breath far in excess of physical signs. The general appearance of the affected miner is that of a man still in health. He is frequently slightly bronzed and looks weather-beaten, and yet the disease may already have got a good grip of his lungs for on physical examination the chest is observed to expand feebly, there is a diminution of 1 in. or more in the ratio between inspiratory and expiratory measurements, also impairment of the percussive note at the base of the lung toward the axillary area with deficient respiratory murmur. The pulse is almost invariably quickened. In the later stages the respiratory murmur may become coarser,

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crepitation may be heard, also a cardio-respiratory murmur along the left border of the cardiac area. The expectoration varies. In the early stages if there is little bronchial catarrh expectoration may be scanty, stringy and pearly white in character; in the later stages it may be mucopurulent, bluish black and rich in particles of silica. In the last stage of the disease it frequently contains tubercle bacilli. As the disease proceeds the tendency is for the expansive power of the chest still further to diminish in consequence of pleuritic adhesion and an increasing loss of elasticity of the lung due to extension of the fibrosis. Although gold miners' phthisis is of slow development, men have died suddenly from cardiac failure when at work in the mine. In only a few of the patients is hemoptysis a symptom. The thoracic organs are those which alone suffer. Those of the abdomen remain healthy; the urine is free from albumen. In the hands of some medical men the application of the Röntgen rays for illuminating the chest has been of service in showing the extent to which the lungs are infiltrated with dust.

Prognosis.—The prognosis is good only in the early stages, provided the patient gives up work and takes to a life in the open. Once tubercle bacilli have settled in the lung the patient goes rapidly down hill. Almost similar symptoms are observed in workers in other dusty trades. In some, bronchial symptoms predominate. Potters, for example, have been known to exhibit a high mortality rate from lung diseases. Potter's rot or asthma was described by Arlidge and attributed by him to the inhalation of fine flint dust which is, practically speaking, pure silica. The symtpoms are morning cough and shortness of breath with unimpaired general health; later the cough becomes paroxysmal and is attended by a degree of dyspnœa in excess of that observed in phthisis. Blood spitting is unusual. On physical examination of the chest there are patches of dulness detected at the bases and signs of empyema in the front of the chest. Owing to the strain imposed upon the right side of the heart this organ dilates. The malady at first is non-tuberculous, but as time goes on the lungs may become infected by Koch's bacillus.

Metal grinders are especially exposed to the risk of pulmonary disease owing to the hard and angular form of the dust inhaled. Sanfield speaking of Sheffield steel grinders points out that they have a mortality rate from pulmonary phthisis three times greater than that of cutlers. The disease assumes the form of fibrosis and is attended by diminished expansion of the chest. In all of us as age advances the power of expansion of the chest diminishes. At the age period 45–49 years the normal individual expires 419 cc. of air less than he did at the age period 20–24, but A. E. Barnes of Sheffield states that the cutler expires 732 cc. less and the steel grinder 751 cc. less. Collis found a high blood pressure run concurrently with diminished chest expansion.

The Transvaal Chamber of Mines[6] has published in its Reports certain schedules of the Miners' Phthisis Board. In one of these the expectation of

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life based upon the permanently impaired physical capacity of the men for underground work is compared with the normal expectation of life of men of the same age calculated according to the Carlisle tables. The conclusion drawn is that whereas a person of the age of 42⅜ years should have on an average of 26 years to live, a person of the same age who has worked underground in the mines 9½ years would have an expectation of only 1½ years. The following figures, taken at random, for 200 cases of beneficiary miners supplied by the Miners' Phthisis Board for 6 months ending January 31, 1913, are not without interest even if they have not met with general acceptance.

Actual service underground	Average actual service underground, years	Average age of miners in each class, years	Normal expectation of life, Carlisle tables, years	Medical advisers' certificate of expectation of life average, years
8 years or over	9½	42⅜	26	1½
7 to 8 years...	7⅙	36½	31	2⅙
6 to 7 years...	6⅙	38¾	28	2⅜
5 to 6 years...	5¼	36	30	3
4 to 5 years...	4¼	40¾	27	3⅓
2 to 4 years...	3	34	31	3½

Of the miners who applied for compensation under the Miners' Phthisis Act, 1912, and have since died but were examined by the Medical Advisers to the Board before death, the number of deaths was 39. The Medical Advisers' average expectation of the applicants' life was 7⅗ months, instead their actual average life was only 1⅘ months.

Pathology.—Irrespective of the particular kind of dust which has led to the development of pneumokoniosis, one and the same structural change is invariably present in all affected lungs, if only sufficient time is given. I refer to an increase of the fibro-connective tissue of the organs, an interstitial pneumonia in fact, accompanied by the presence of pigment without recognizable evidence of tubercle or syphilis. The fibrous tissue tends to assume in places concentric formation, the fibrosis being particularly well marked around blood-vessels and small bronchi. It is around these that the earliest changes are detected. The blood-vessels become thickened as a result of endarteritis or in consequence of prolonged venous congestion. There are usually signs of bronchial catarrh. On microscopical examination particles of dust can be seen imbedded in large endothelial cells lying inside the alveoli and also in the fibroblasts. Occasionally free red blood cells can be seen occupying the alveoli as a consequence of venous congestion or more probably as a consequence of hemorrhage from newly developed thin-walled vessels such as occur in granulative tissue. The development of new blood-vessels is an interesting feature in pneumokonioses, for while at first they are capable of meeting all nutritive demands the blood supply ultimately becomes restricted, a condition which may lead to necrosis.

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This brief statement requires a further and more carefully detailed account of the microscopical appearance of sections of a lung in pneumokonioses, and as the structural changes are particularly well marked in a non-tuberculous fibrotic lung, which was sent to me by Dr. Aitken of the Miners' Sanitarium, South Africa, I will make these the basis of my remarks. For assistance in the description of the pathological changes I am indebted to my colleague, Prof. Stuart McDonald. When animals have been exposed interruptedly to a dusty atmosphere over a considerable length of time one of the earliest signs in the lungs is the presence of large pigmented cells lying free within the alveoli. These cells are phagocytes. Some of the cells may be extended leucocytes, others are modified alveolar endothelial cells. During the act of coughing some of these pigmented cells as well as others lying on the bronchial mucous membrane are expelled and can be found in the expectoration. In another way the fate of these dust alveoli is interesting. Many of them penetrate through the stomata, they break through between the alveolar epithelium into the lymphatics of the fibro-connective tissue framework of the lung and are carried to the glands at the hilus or they disintegrate and induce an irritative fibrous hyperplasia. Irritation by this foreign material is capable alone of inducing the fibrous overgrowth observed in typical silicotic nodules, but probably other factors are in operation as well. Many of the smaller bronchioles have their lumen blocked by catarrhal products and by œdema attended by collapse of the immediate lung tissue. Where collapse has occurred, the alveolar walls become thickened and there develops an area of fibrous tissue which is, practically speaking, free from dust particles. Here and there may be seen small areas of lung, seats of acute pneumonic consolidation. The intra-alveolar exudate varies. It may be composed of mononuclear cells which have come from a proliferating alveolar endothelium, or it may be fibrinous in character and include numerous leucocytes within its meshes. Numerous free red blood cells are seen inside the alveoli and in irregular situations. The presence of newly formed capillaries in the exudate points to a fibrous organization taking place such as is occasionally seen in an unresolved pneumonia. As regards the source of the newly formed fibrous tissue it may be that some of the larger mononuclear cells and also some of the smaller cells of the lymphocyte type play a part. The bulk of it is the product of fibroblastic cells, which, spindle-shaped, flattened or branch-like, can be seen at the periphery of a densely fibrotic silicotic nodule while the smaller lymphocyte cells are situated more externally. The fibroblasts are also phagocytic for several of them can be seen ladened with foreign particles. Branches of the pulmonary artery show fibrous and elastic thickening of their inner coat. This is not so apparent in the finer divisions of the pulmonary vein. The alveolar capillaries are overfilled in places and antemortem thrombosis of the smaller blood-vessels is here and there apparent. Scattered through the lung are dense silicotic nodules. Most of these are avascular but where the morbid change is still

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progressing the tissue is distinctly vascular. In none of the sections neither to the naked eye nor on microscopical examination is there the slightest evidence of tubercle. Where the changes have become most marked all traces of alveolar structure have disappeared; the outline of thickened and obliterated vessels may still be traced amid accumulations of dust particles, while as regards connective-tissue corpuscles these are represented by little else than free nuclei. The absence of pus cells is a notable feature. The presence of pus corpuscles is usually a sign that tubercle has become grafted upon a lesion which was primarily non-tuberculous. In a large percentage of the cases, probably 70, death comes by secondary tuberculosis. On careful examination of the structureless material of fibrotic tissue and amid mineral particles tubercle bacilli may be found, but if present they are few in number and are without their usual histological accompaniment, giant cells.

In the lung of a dog, the subject of experimental anthracosis, I was surprised at the large size of the particles of coal dust found in the pulmonary alveoli after death. The transport of dust into the lungs during inspiration is a fact beyond controversy—notwithstanding all that has been written upon the subject of tidal air in respiration and the renewal of the air in the small bronchi and pulmonary alveoli by diffusion. During inspiration when men are working hard a portion of the tidal air must be drawn into the pulmonary alveoli.

Silicosis and Tuberculosis.—Since in a considerable number of persons dying from silicosis no sign of tubercle is found in the lungs or in the body generally, silicosis must therefore in its early stages at least be regarded as a non-tuberculous disease. In some of my own patients the malady from first to last was non-tuberculous. When tuberculosis develops not only is it secondary to pneumokoniosis but there occurs an alteration in the symptoms. Pneumokoniosis is of gradual development; it usually pursues its cause without any pronounced rise of temperature. Once tubercle has been grafted upon the malady, emaciation becomes progressive, the temperature rises, there are night sweats, the expectoration becomes mucopurulent and frequently contains Koch's bacillus. Experience confirms the opinion based upon experimentally produced dust diseases of the lungs, viz., that a patient suffering from pneumokoniosis is more prone to develop tuberculosis than a person otherwise healthy. It might appear as if the avascular state of the fibrotic areas of an affected lung did not lend itself to the reception and multiplication of the tubercle bacillus, but a suitable nidus is certainly provided in the catarrhal secretion within the pulmonary alveoli. That dust diseases of the lung specially predispose to other microbic diseases such as pneumonia is the experience of medical men on the Rand. The Kaffirs employed in the gold mines round Johannesburg have an extremely high death rate from pneumonia. Not only do the native miners take the disease readily, but their resistance to it is diminished.

The South African Miners' Phthisis Commission of 1912 reported that

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silicotic lungs become increasingly liable to infection as the fibrosis progresses. Of the patients in the early stage of the disease 6 per cent. were found to be tuberculous; of those in the middle stage 12 per cent. and in the later stages 44 per cent.

Dr. G. A. Turner of Johannesburg and Dr. D. Macaulay have drawn attention to a form of tuberuclosis affecting the spleen and mesenteric gland, found in gold miners. Not only is the spleen studded with nodules, but in the glands there is observed proliferation of the proper elements with fibrosis, and signs of tubercle bacilli are present in small numbers and are with difficulty detected. As indicating the types of tuberculosis found in the Kaffirs employed in the gold mines, Dr. Turner reports that of 309 deaths which occurred in one of the Compound Hospitals during the 6 years 1908–1913 and which were confirmed by post-mortem examination, 140 were pulmonary, 92 were cases of generalized tuberculosis, 33 peritoneal, 33 splenic, 7 hepatic, 3 meningeal and 1 renal. Dr. G. D. Maynard[7] also found tuberculous abdominal lesions prevalent in native miners and is therefore disposed to admit the possibility of infection having occurred through the alimentary canal. When we remember that in the mines the expectoration of the men who are the subjects of the malady is widely distributed and is found on the walls, floors, steps, hand rails of ladders, on the inside of cages, also that the native miners eat their food with unwashed hands, infection by the alimentary canal is more than a possibility. These remarks raise the question as to what is the mode of entrance of dust into the lungs?

Mode of Entrance of Dust into the Lungs

Is pulmonary anthracosis of aerial or intestinal origin? Where men had been working in a dusty atmosphere and their lungs after death are found to be deeply pigmented it is natural to suppose that the altered condition of the lungs is the result of particles of dust which had been inhaled when at work. Physical and chemical confirmation of the identity of the dust found in the lungs with that present in the atmosphere of the workshop lends weight to this supposition. Calmette of Lille, Van Steenberghe and Grysez in their researches upon tubercle concluded that tuberculous lesions of the lungs are not so much the consequences of aerial infection by Koch's bacillus as that the microörganism enters the body by the alimentary canal and finally reaches the lungs by the lymphatics. If, as these pathologists maintain, this is the channel of infection by tubercle bacilli, so in all probability is it the portal of entry of the particles of carbon which cause the anthracosis of coal miners. In their experiments they found that the ultimate destination of pigment was greatly influenced by the age of the animal experimented upon. In the lungs of young guinea-pigs particles of carbon were not found, but they were present in the lungs of older animals, the opinion being that while carbon dust was arrested in the mesenteric and other abdominal glands of younger

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animals the more open meshes of the glandular structure in older animals permitted the passage of the particles through the glands, and thus allowed of their transport by the lymphatics to the lungs. I have carried out several experiments bearing upon the artificial production of anthracosis both by the intestinal canal and the respiratory passages. My own experience is that while pigmentation of the lung can be produced by materials carried into the intestinal canal, there is no comparison between this as the portal of entry and respiratory passages. The presence of particles of carbon lying free within the the pulmonary alveoli and their presence too in large phagocytic cells lying loose in the alveoli indicate that these particles must have reached the lung by the air channels. The absence of pigment in the lungs of animals whose esophagus had been ligatured and which had breathed air in which particles of soot were freely suspended is admittedly strong evidence in support of the intestinal mode of entrance as advanced by Calmette and his school, so too his other experiment where pigment was found in the lungs when the esophagus was open but one of the bronchi ligatured. In Great Britain Professor Whitla of Belfast is one of the strongest adherents of the intestinal origin of anthracosis. His conclusions rest upon data obtained by repeating Calmette's experiments and especially those of the introduction of Indian ink by the intraperitoneal method. In my own intraperitoneal experiments I have been impressed by the rapidity with which even considerable particles of coal dust were transported to the abdominal glands, including such organs as the testes. The peritoneal cavity is a large lymphatic space from which infective material is rapidly absorbed and conveyed by the lymphatics, but although such material may finally reach the lungs it never reaches them in such quantity as when aspirated into the lungs by breathing. Supporters of the theory of the intestinal origin of pulmonary anthracosis lay stress upon the mechanical obstacles such as the presence of ciliated epithelium in the upper portions of the respiratory canal, the branching and narrowing of the bronchial tubes which render it difficult for dust to reach the lungs. The abdominal glands of coal miners are occasionally black but never in comparison with the bronchial glands or the lungs. In the alveoli of the lungs of rabbits and dogs exposed for 1 hour daily to an atmosphere rendered dusty by means of finely powdered coal there can be seen numerous large cells of the epithelioid type which have engulfed fine particles of dust. These cells are phagocytes. They are expelled in the expectoration or they may penetrate through the alveolar endothelium and reach the connective-tissue framework of the lungs or the lymphatics. They are carried by the lymphatics to the bronchial glands where they unburden themselves of the pigment they have brought or they undergo disintegration dispersing the pigment as fine dust. Instead of using particles of soot or of Indian ink, iron carbonate can be administered and its presence subsequently demonstrated by a blue color obtained with hydrochloric acid and ferrocyanide of potassium. Dr. Leonard Findlay[8] repeated Calmette's experiments and he found

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that where animals were fed for days with emulsions of soot or Indian ink through a stomach tube the lungs showed no abnormal pigmentation, that when fed with iron carbonate by the stomach tube for 27 days, while large quantities of iron carbonate were present in the mesenteric glands, the lungs, with the exception of a small patch of broncho-pneumonia, were free from iron carbonate although particles of soot were present in the epithelial cells of the alveoli. Where animals breathed an atmosphere ladened with soot, the esophagus and both of the bronchi at the time being free, the lungs were found invariably pigmented; in the case of other animals breathing a similar atmosphere but whose esophagus had been ligatured the lungs were pigmented and the alveolar cells contained carbon, but the mesenteric glands were free from soot. Finlay caused a tracheotomized animal to breathe a soot-ladened atmosphere, its left bronchus being plugged with cotton wool. The animal inhaled dust for 2 hours. When killed the right lung was of dark color while the left still retained the rosy appearance of health. In the alveoli of the right lung were found considerable amounts of pigment. Where animals breathed for varying periods iron carbonate, their esophagus and bronchi free, the alveolar cells gave the Prussian blue reaction with potassium ferrocyanide. After intraperitoneal injections of soot, also of Indian ink, the lungs were usually entirely free from pigment while the mesenteric glands were pigmented. In a few instances the mediastinal glands were pigmented and the lymphatics of the lungs which run in the alveolar walls contained the colored material. It is difficult to produce pulmonary anthracosis by feeding. The difference in the pigmentation of lung caused by the inhalation of foreign particles and the same when introduced by the alimentary canal is that when dust has been inhaled the dust particles are found lying inside the pulmonary alveoli, whereas when introduced by the intestinal canal if they reach the lung at all they are found lying in the interior of the lymphatics. My own opinion in regard to the origin of pulmonary anthracosis is that while the dust may reach the lungs by both the intestinal ahd respiratory passages the latter is the more frequent of the two.

Prof. Beattie of Liverpool University has been extremely successful in producing the early stages of pneumokoniosis in guinea-pigs by causing them to breathe a dusty atmosphere. Sections of the affected lungs show the thickened alveolar walls due to proliferation of fibro-connective tissue, the presence of pigment in the pulmonary lymphatics also, in the large phagocytic cells lying within the pulmonary alveoli. His experiments prove that prolonged inhalation of dust is capable of producing structural alterations similar to those found in the lungs of miners dying from uncomplicated pneumokoniosis.

Treatment.—If pneumokoniosis cannot be entirely abolished, it certainly can be largely prevented. It is a matter of getting rid of the dust to which men are exposed when at work, whether in the factory, grinding shed, quarry

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or mine. The improved conditions under which coal miners work, their better housing, shorter hours and higher wages, but above all the freer ventilation of coal mines by larger fans and by double shafts for "intake" and "outgo" air, have considerably reduced the mortality rate of miners' phthisis. Ventilation of coal mines, however, has its limits. There is just the risk of excessive ventilation sweeping away from the walls, floors, roofs and props and carrying forward through a mine the extremely fine dust which lies in the mainways. This fine dust is not only a menace to the health of the miners but a cause of explosion in coal mines. Spraying with water has, therefore, been resorted to, but coal dust and water do not mix together readily. Coal dust floats in water and is not quickly permeated by it; besides coal dust which has been wetted with water on becoming dry is found to have undergone further disintegration and to have become a finer form of dust. Soap-and-water spraying and spraying by other fluids have been recommended since they bind the dust and render it difficult for currents of air to lift it. Herman Belger of the Armstrong College, Newcastle-upon-Tyne, has for the last 2 years carried out in my laboratory at the College of Medicine a series of experiments as to how to bind the dust so that it will remain bound under all conditions of temperature and dryness of the atmosphere. His solution possesses the property of abstracting moisture from the atmosphere so that a dusty surface treated by it remains more or less moist and therefore attractive to fresh dust. Belger's solution is a colloid and contains calcium and other salts. The colloid solution is harmless from a health point of view; it has no injurious influence upon the plant of a mine or upon the seam of coal rock or ore. Spraying of the interior of coal mines by Belger's solution has been tried over several miles of roadway in coal mines with great success. The colloid solution not only renders a mine free from dust but keeps it free. Wherever it has been sprayed there is created a coolness in the underground passages which makes the work of the miner easier, and at the same time it diminishes the risk of explosion.

There are dusty occupations in which men can work wearing a respirator. Inhalation of dust is thereby to a great extent diminished, but where the work is hard and of a laborious nature men complain that they cannot wear a respirator. Theoretically the wearing of a respirator is all right, but it creates heat and the men cannot breathe freely through the respirator.

Of the curative treatment of pneumokoniosis little can be said. It is only in the early stages that satisfactory results can be obtained, and that is by the men on the first approach of symptoms retiring from the work and taking up occupations in the open air. I know of no specific medicinal treatment for pneumokoniosis. Once fibrosis has begun there is always the probability of it progressing. If, however, the patient can be kept free from the possibility of tuberculous infection he may live for years, provided he is careful in his habits and avoids catching cold. By the administration of calcium salts such as chloride combined with a tonic like nux vomica, good

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food, abstinence from alcohol and by living in the open air health can be well maintained and a comparatively useful life spent. Once tubercle bacilli invade the lung the patient rapidly becomes worse and the case practically becomes hopeless. Silicosis and tuberculosis are mutually destructive to each other. A silicotic lung soon breaks up in the presence of tubercle bacilli.

On the question of the treatment of silicosis it would be well to read what Dr. Francis Aitken[9] has to say, since he has had experience which falls to few physicians. In November, 1911, there was opened at Modderfontein, South Africa, a sanatorium for the treatment of miners' phthisis. Dr. Aitken, Medical Superintendent, states that since the opening of the sanatorium, the end of December, 1913, there have been admitted 322 cases. Miners in all stages of the disease, with or without tuberculosis, have been received. Forty per cent. of the men admitted were tuberculous and had to be segregated from the other inmates. During 1913, 156 patients entered the sanatorium; 103 were discharged and 48 died. In regard to the curability of silicosis the Medical Superintendent expresses himself thus: "Recent researches in the pathology of the disease reveal the important fact that the fibrous tissue which is formed as a result of the irritation by the dust in the lungs, and which is responsible for the hardening of the lungs and their impairment as respiratory organs, tends in time to become organized and permeated by newly formed blood-vessels. As a result, the fibrous tissue is in part absorbed and the particles of silica are liberated and carried away in the blood stream. This process makes for a good prognosis in cases which (1) are no longer exposed to a dusty atmosphere, (2) are free from tuberculosis, (3) have no breaking down of the lungs, (4) are able to survive the period of heart strain that always accompanies any marked degree of silicosis. Thus it would appear that early cases of silicosis that are able to fulfil these conditions should be regarded as curable and more advanced cases should improve to a marked extent." Without comment, I close this paper with the above expression of optimism on the part of Dr. Aitken.

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CHAPTER II

DISEASES OF THE BLOOD, CIRCULATORY SYSTEM AND KIDNEYS

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As this chapter must of necessity be limited in its scope, only the main features of the diseases can be considered. For convenience these diseases will be treated under their usual headings and special attention given to the facts that bear more or less directly on the occupational aspects of the subject.

Brief mention is made of the trades or occcupations, in which statistics show that these diseases are unusually frequent, a fuller consideration of the different occupations being found under their separate headings in another section of the book.

The etiological factors considered being only those unavoidably connected with the various industries, no mention is made of the effects of alcohol and syphilis which, though unusually prevalent among certain classes of workers, are in no way necessarily connected with the occupations themselves.

Broadly speaking, the etiological factors of the diseases of the blood, the circulatory system and the kidneys, considered as occupational diseases, are either those that act mechanically, such as strain and exposure to physical forces, or the industrial chemical poisons. The defective hygienic surroundings under which, almost of necessity, many trades are carried on also have their part in causing certain of these diseases.

DISEASES OF THE BLOOD

Under the heading of Diseases of the Blood will be considered the secondary or symptomatic anæmias, as well as certain pathological conditions of the blood that are brought about by industrial poisons. These latter are not in themselves disease entities, but are commonly important features of the poisoning and not infrequently dominate the picture of the syndrome. A full account of the more important poisons is given in the preceding chapters.

Hemoglobinemia.—The abnormal presence of hemoglobin in the plasma of the blood is brought about by hemolysis. In this manner the hemoglobin is discharged from the erythrocytes, and to a large extent they are destroyed. Among the industrial poisons causing hemolysis may be mentioned arseniuretted hydrogen and carburetted hydrogen, also phosphuretted hy-

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drogen which together with arseniuretted hydrogen is liberated when ferrosilicon comes into contact with water. Many cases have been reported in recent years in ships carrying ferrosilicon. If, as often happens, the hemolysis is rapid and overwhelming, a fatal result ensues with tissue suffocation. If less severe, the patient presents the typical symptoms of acute secondary anæmia, whereas a moderate hemolysis acting over a longer period of time is one of the recognized causes of chronic secondary anæmia.

Methemoglobinemia.—Methemoglobin is found partly in the erythrocytes and partly in the plasma as the result of an associated hemolysis. It is considered isomeric with oxyhemoglobin, but is a more stable compound and is not capable of carrying on its oxygenating functions. It imparts a brownish or chocolate color to the blood and is easily recognizable by its characteristic spectrum. Among the industrial poisons causing methemoglobinemia may be mentioned benzene, nitrobenzol, nitrous gases, phenylhydrazine, aniline, and various aniline dye stuffs. Many other substances may bring about this condition, but they appear to be of greater medical or chemical than industrial interest.

Sulphemoglobinemia.—Sulphemoglobin is formed by the action of sulphuretted hydrogen on the blood, and it has been found that in the presence of a reducing or oxidizing agent very small quantities may bring about this change. It gives the blood a purple color and has its characteristic spectrum, slightly different from that of methemoglobin. The first cases were reported by Van der Bergh in 1905. These were idiopathic or enterogenous. A number of cases have been reported since, both of this type and following industrial poisoning.

Symptomatology of Methemoglobinemia and Sulphemoglobinemia.—Large doses of some of the poisons causing these disturbances, as for example the breathing of air charged with a high proportion of sulphuretted hydrogen, are rapidly fatal. Death may be preceded by a short stage of unconsciousness, with slow pulse, Cheyne-Stokes respiration and increasing asphyxia—symptoms doubtless due to bulbar paralysis. In the more prolonged and non-fatal cases marked cyanosis develops—a gray-blue discoloration of the skin and mucous membranes. Headache with gastric disturbances and general muscular weakness are associated symptoms. In the enterogenous cases, more particularly, it has been noted that chronic diarrhea is present in methemoglobinemia and constipation in sulphemoglobinemia. The blood may not show great abnormality on ordinary examination, a moderate degree of anæmia being present in the acute cases. Spectroscopic examination is pathognomonic.

Carboxyhemoglobinemia.—This is typical of carbon monoxide poisoning. The blood is bright cherry-red in color, flows very freely, and presents a definite spectrum. Carbon monoxide has an affinity for hemoglobin more than 200 times greater than that of oxygen, so that even small amounts in the inhaled air are readily absorbed by the blood and retained

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in stable combination. Carboxyhemoglobin does not react with reducing agents and is useless for purposes of oxygenation. It has been found by experiment that the effects of the poisoning are not noticed until about one-third of the hemoglobin has been converted into carboxyhemoglobin, that they become urgent when the corpuscles are about half saturated and that death usually occurs when the respiratory capacity of the blood has been reduced to about 30 per cent. of the normal. A leucocytosis is sometimes present and in the chronic cases an increase in the number of the erythrocytes.

Symptomatology.—The symptoms are mainly due to the changes in the blood, and defective internal respiration. In a highly contaminated atmosphere the attack may be very sudden. The person falls unconscious with loss of muscular power, and the heart's action, becoming progressively weaker, soon stops. Usually the symptoms of acute poisoning are gradual in their onset—first severe frontal headache, throbbing of the arteries, buzzing in the ears, vertigo, nausea and vomiting, muscular weakness, dyspnœa and oppression, with rapid pulse and respirations. Pale or bright red patches are seen upon the skin due to local dilatation of the peripheral vessels and the change in the color of the blood. Sometimes, however, a bluish discoloration of the skin is more noticeable. Later there are motor disturbances, tremors, convulsions and paralyses with loss of consciousness. Death is usually due to respiratory paralysis and asphyxia; or recovery may occur even after severe poisoning, with a slow convalescence.

Chronic poisoning is characterized by headache, dizziness, nausea, impairment of memory, muscular weakness and secondary anæmia.

Cyanmethemoglobinemia is present in poisoning by the cyanogen compounds, especially by the fumes of hydrocyanic acid and the cyanides. The cyanogen ion acts both upon the hemoglobin, forming a new compound with characteristic spectrum, and upon the tissues, directly inhibiting their power of oxygen absorption. The result is tissue asphyxia. The blood is a bright red color and the venous return flow resembles the arterial. The alkalinity of the blood is diminished and lactic acid appears therein.

Symptomatology.—Death follows quickly on the inhalation of large quantities. It may be preceded by a stage of deep unconsciousness, with dyspnœa and heart failure. In the acute but non-fatal cases there is headache, vertigo, palpitation, dyspnœa and great prostration. Convulsions and loss of consciousness may follow. There is usually cyanosis of the skin and mucous membranes. In those who are exposed to constant inhalations of minute quantities of the vapor, a form of chronic poisoning may develop, in which all of the above symptoms are present in a minor degree. Anorexia, nausea and disturbance of the gastro-intestinal functions are also present, and the picture of a chronic secondary anæmia is presented.

Acute Secondary Anæmia.—As an occupational disease, this is caused by acute intoxication by various industrial poisons. These comprise for the most part those mentioned in the previous section, and in many cases other

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signs of acute anæmia are added to the changes in the hemoglobin. Acute anæmia may also be caused by other poisons, such as carbon disulphide or mercury, which cause rapid destruction of the elements of the blood.

Symptomatology.—The condition is often ushered in with nausea and vomiting. Dyspnœa is an early and constant symptom and there is usually a feeling of anxiety and extreme difficulty in breathing. With this is associated faintness, vertigo and a small rapid pulse and subnormal temperature. Headache and severe nervous symptoms are frequently present. There is marked pallor of the skin except in those cases where the hemoglobin is destroyed, when we have the various characteristic changes previously noted. The blood picture may be that of chronic secondary anæmia, described in the next section, but often resembles the pernicious type—great reduction in erythrocytes with a relatively high hemoglobin content and high color index, extreme variations in shape and size of the erythrocytes with an excess of oversized cells, polychromatophilia, many normoblasts and megaloblasts. A leucocytosis is, however, usually present, except in the case of benzol poisoning, where there is a marked leucopenia.

Treatment.—Oxygen is our most valuable agent in combating the sudden and extreme diminution of the hemoglobin content of the blood. Oxygen treatment, as stated by Rambousek, "rests on the fact that by raising the pressure of the oxygen from 113 mm., as it is generally in ordinary air, to 675 mm., which is reached in the presence of pure oxygen, the quantity of oxygen absorbed by the blood rises from 0.3 to 1.8 per 100 c.c. Further, the saturation of the hemoglobin undergoes an increase of 2.4 per cent." This increase of oxygen in the blood, he says, can save life in cases where, through poisoning, a deficiency of oxygen has resulted. Special apparatus for the administration of oxygen should be provided wherever there is a likelihood of industrial blood poisoning. In acute hemoglobin transformation venesection with infusion of normal saline or direct transfusion of blood is indicated. This relieves the blood of at least a part of its useless hemoglobin compound and remaining toxin, and may just suffice to enable the organism to turn the scale in its efforts for regeneration.

During convalescence the case should be treated as one of chronic secondary anæmia.

Chronic Secondary Anæmia.—This disease may be due to the effects of the insanitary conditions that necessarily surround certain occupations. Bad air, prolonged exposure to heat, frequent and sudden changes of temperature, and irregularity in eating all tend to bring about a condition of inanition in which a typical secondary anæmia plays the conspicuous part. Statistics show that this is unusually prevalent among bakers, iron and foundry workers, miners and smiths. Less typical forms of chronic secondary anæmia, characterized in some cases by special features, are caused by the continued or frequent action of poisons to which industrial workers are accidentally or necessarily exposed. Among these poisons may be men-

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tioned lead, mercury, antimony (which causes also leucopenia and eosinophilia), phosphorus, chlorine and bromine, as also the dust inhaled in the cotton and jute industries. Many of the poisons causing acute anæmia may also cause the chronic form, if they act in smaller quantities over a longer period of time.

Symptomatology.—Pallor of the skin and mucous membranes is marked. There is loss of weight, with muscular weakness and loss of mental vigor. The appetite is poor, the digestion impaired, the nourishment fails. There is usually palpitation, an irritable heart, rapid pulse, with feelings of faintness and vertigo, and, later, signs of inefficient circulation, such as constant fatigue, rapid respiration, dyspnœa on exertion, and swelling of the feet. There may be extravasations of blood into the mucous membranes, petechiæ in the skin, or retinal hemorrhages. There is frequently a slight remittent fever.

The blood shows a moderate reduction in the number of erythrocytes to between 2,000,000 and 4,000,000 per cu. mm., greater proportionate reduction of hemoglobin, giving a color index of about 0.8 to 0.5; many erythrocytes are pale, irregular, nucleated, polychromatophilic; the leucocytes are slightly increased in number, usually the multinuclear neutrophiles.

In chronic hemolysis the blood approaches the type of pernicious anæmia: a great reduction in the number of erythrocytes, which frequently fall below 2,000,000 per cu. mm., great variations in size with a general average increase, and a relatively high hemoglobin content, giving a high color index, and the presence of many normoblasts and megaloblasts.

In chronic lead poisoning many of the erythrocytes are small and with basic stains exhibit a sprinkling of blue-black dots upon the corpuscle, known as stippling or basophilic granulation. This may be associated with the blood picture of pernicious anæmia, but often it is accompanied by but few other abnormal signs.

Treatment.—But little can be done in the way of treatment without removal of the cause. The patient must be placed in good hygienic surroundings and the poison eliminated where possible. This frequently necessitates a change of occupation. Fresh air and a nutritious diet are the main points to be insisted upon. Iron in any form and in full doses is indicated, with arsenic as an adjuvant. Care must be taken to avoid constipation. In the severe cases rest in bed is necessary for a certain period.

DISEASES OF THE CIRCULATORY SYSTEM

Primary Cardiac Overstrain.—Although many, perhaps most, of the persons who are subject to attacks of cardiac failure with dilatation are possessors of a damaged myocardium, most authors now agree that disturbance, inefficiency or failure of the circulation may be brought about in a healthy heart by overstrain. As an occupational disease, primary cardiac

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overstrain is frequent among soldiers, porters, miners, blacksmiths and metal workers.

Pathology.—The cavities of the heart are usually dilated and the muscular walls frequently show a certain amount of antecedent hypertrophy, without degeneration. The muscular tonicity is diminished, but otherwise the heart is normal.

Symptomatology.—The cardinal symptom is a limitation of the field of cardiac response, the patient being unable to support the least exertion without distress, though he may be comfortable when at rest. There is frequently palpitation, cardiac pain, a sense of pressure or constriction over the chest, often restlessness, headache, buzzing in the ears and vertigo. The pulse is small, feeble and rapid, and sometimes irregular; the cardiac impulse is wavy and diffused; the area of dullness enlarged; the sounds distant and feeble, or short and sharp; and a soft blowing systolic murmur of functional mitral or tricuspid insufficiency is often present. In the more severe cases, or with those who make an effort to continue their work, dyspnœa, œdema, and other symptoms of cardiac decompensation ensue.

Treatment.—For the sake of emphasis the treatment might almost be summed up in one word—prolonged rest. Some weeks or months are none too long in a moderately severe case. This must of course be supplemented by adjuvant measures as to hygiene, diet, etc. Moderate doses of digitalis, 1 to 2 grains a day, are a great aid in restoring the tonicity of the heart muscle. The return from complete rest to full activity must be made very gradually. What can be accomplished by care and prolonged treatment is shown by the remarkable results obtained by da Costa in many very severe cases among Union soldiers of the Civil War. The fact that many of these were again able to perform strenuous work and lead perfectly healthy lives is further proof, it any were needed, that cardiac overstrain and dilatation may be a primary disease.

Functional Affections of the Heart.—Recent advances in cardiac pathology have tended to limit considerably the field of purely functional affections of the heart. It has beeen shown that many diseases previously considered functional are in reality due to lesions of some part of the cardiac musculature. It is still customary and convenient, however, to group under this heading various disturbances of the rate, rhythm or force of the heart's action. Of these, palpitation, tachycardia, and bradycardia are frequently caused by the chemical industrial poisons and may be the chief manifestation of such poisoning, but more often are only a part of the general symptom-complex.

Palpitation is the forcible overaction of the heart, whereby its beating becomes disagreeably perceptible to the individual. It may or may not be associated with some form of irregularity, although it must be emphasized that the great majority of the arrhythmias are now thought to be due to myocardial diseases or degeneration. The industrial poisons most likely

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to cause palpitation are amyl acetate, carbon monoxide, naphtha and gasoline, hydrocyanic acid and the cyanides, and tobacco inhaled as dust in the process of manufacture.

The term tachycardia is usually applied to a condition of marked rapidity of the heart's action, independent of fever or evident physical cause such as exercise or excitement. It may be continuous or intermittent and the rhythm may be regular or irregular. Its etiology is varied, the more common forms being due to heterogenetic impulse formation, and being designated according to their nature as auricular flutter, auricular fibrillation, essential paroxysmal tachycardia. More rarely the cause is a destructive lesion of the pneumogastric nerve. These forms must be differentiated from the tachycardia due to chemical poisoning. As an industrial disturbance this is usually associated with poisoning by acetaldehyde, aniline and the nitrophenols.

Bradycardia indicates a condition in which the heart's action is abnormally slow, although here it is difficult to set the limits between health and disease. Although some people are said to have had healthy lives with extremely slow hearts, a pulse below 50 must practically always indicate some disturbance either organic or functional. Except in the terminal stages of disease, a pulse below 35 is usually due to heart-block, while one between 35 and 50 may result from many causes. Bradycardia is more frequent than palpitation or tachycardia in the severe cases of chemical poisoning, and is a usual accompaniment of poisoning by carbon monoxide, hydrocyanic acid and the cyanides and sulphuretted hydrogen.

Pathology.—These functional cardiac disturbances in chemical poisoning are due to the direct action of the poisons on the heart or its nervous mechanism, or to the blood changes which they produce. In one or the other manner the cardiac musculature may be deprived of its proper nutrition and rendered inefficient; or the inner stimulus of the blood on which the heart depends for its automatic rhythmicity may be disorganized; or the centers or terminals of the vagus or accelerator nerves may be either irritated or depressed, according to the nature or selective action of the poison.

Treatment.—The first object to be attained in the treatment of these toxic conditions is the elimination of the poison, hence free catharsis and diuresis are always indicated, and in the acute cases chemical antidotes where possible. Rest to a considerable degree is called for, although the more nearly the disturbance approaches the purely nervous type, the less is this necessary or advisable. Here well-regulated exercise may often be substituted.

For palpitation and tachycardia the bromides of potassium, ammonium and strontium are of some value, and large doses of aconite sometimes act favorably. The application of an ice-bag or a cold-water coil over the heart is often the best remedy in the acute cases. If the bradycardia is due, as is often the case, mainly to increased inhibition, atropine is indicated for its selective action on the vagal terminals. Here, however, we

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are more likely to have some signs of circulatory inefficiency and must often have resort to caffeine or digitalis.

Acute Endocarditis.—Acute endocarditis usually follows or is one of the manifestations of acute rheumatic fever, and is frequently met with as an occupational disease in accordance with the prevalence of the latter among certain workers. Although this form of rheumatism is now generally conceded to be due to a specific organism, the infecting agent is doubtless widespread, and certainly attacks with greater frequency those exposed to severe cold or dampness, and especially to sudden changes of temperature. The continued inhalation of certain forms of dust seems also to be a predisposing cause. These conditions are the necessary accompaniment of certain occupations, and statistics show that acute rheumatism in its arthritic, tonsillar, or endocarditic manifestations is unusually frequent among cotton spinners, wool sorters and drapers, brewers, bakers, carpenters and stokers. Rheumatic endocartitis, therefore, is an increased risk attending these occupations.

Pathology.—Its pathology here in no way differs from that seen in other cases; the arrest of the organisms upon, or more often within, the tissue of the valve, or on the lining membrane of the heart chambers, the deposition of blood platelets, leucocytes and fibrin in varying proportions, forming minute vegetations of a warty or verrucose appearance; later, the organization of these vegetations by the disintegration and absorption of the blood cells and fibrin, and the outgrowth of connective tissue, leading ultimately to sclerosis, thickening and deformity of the valves.

Symptomatology.—The symptomatology of acute rheumatic or simple endocarditis is not very characteristic. There is usually a slight fever, or an exacerbation of a previously existing fever, often palpitation and a pulse rate out of proportion to the fever, usually some respiratory distress, with dyspnœa on exertion, occasionally slight œdema, ascites or enlargement of the liver. Anæmia and anorexia are often associated symptoms. The physical signs are uncertain. There is usually accentuation and sometimes reduplication of the pulmonic second sound, and a systolic bruit at the apex. The latter, however, is more often a sign of myocarditis than of endocarditis, and entirely disappears with the subsidence of the acute attack. Many cases are latent, and the condition is unsuspected, only to become apparent later as a chronic endocarditis.

Treatment.—The treatment of acute rheumatic endocarditis should be mainly prophylactic, and our present knowledge of the subject seems to indicate that tonsillectomy should be considered in all patients who have had rheumatic infections in any of its forms, or who seem to be specially exposed to, or susceptible to it. The further prophylactic treatment consists in prolonged rest during an acute rheumatic infection. This is more potent than salicylates or other specific treatment in decreasing the chances of endocarditis. If endocarditis develops, the rest should be still more prolonged and the active treatment symptomatic.

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Chronic Endocarditis.—This may be a sequel of acute endocarditis; but more frequent, as an occupational disease, is the sclerotic type, of insidious onset and gradual development, which is usually but one manifestation of a more or less generalized sclerosis affecting the heart, arteries and kidneys, and often the liver and spleen. Long-continued overexertion or frequently repeated severe muscular strain are fundamental causes of these lesions, and to these may be added the effects of chronic lead poisoning and chronic anæmia. Among the other causes that are common among industrial workers are gout and intestinal toxæmia due to the prolonged use of an improper diet. An abnormally high percentage of chronic heart cases are found among iron and metal workers who wield heavy hammers and carry heavy burdens in foundries, blacksmiths, miners, bakers, soldiers, and lead workers.

Pathology.—The pathology of chronic endocarditis may be briefly summarized as follows: sometimes as a sequence of an acute endocarditis, more often as a slow and insidious growth as a result of the causes above referred to, the edges of the valves become thickened and present small nodular prominences. The substance of the valve loses its translucency and its delicate tenuity. Later there is a further overgrowth of fibrous tissue, causing marked thickening of the valves or mural endocardium or both. The chordæ tendinæ also are frequently affected. The fibrous tissue then contracts and produces shortening of the chordæ and gross deformity of the valvular segments, causing either a narrowing of the orifice, or a retraction and incompetency of the valves, or both. Finally lime salts are frequently deposited in the sclerotic tissue, so that the remnants of the valve and the orificial ring may become a calcareous mass, quite ineffectual for serving its natural function.

Symptomatology.—The symptoms of chronic endocarditis are those of chronic valvular disease in general. When the heart's hypertrophy and its muscular integrity keep pace with the disability due to the valvular defect, they are but slight, and are evidenced chiefly by a limitation of the field of cardiac response. If this is overstepped, as it almost necessarily will be at an early stage in the patients under consideration, there will be some dyspnœa and general weakness, often with palpitation and precordial distress. As decompensation sets in gradually, or often in more or less sudden and recurring attacks, a train of symptoms is presented due to (1) the slowing of the circulation with diminished supply of oxygen and accumulation of carbon dioxide, (2) the overfilling of the veins with blood, and (3) reflex nervous disturbances. The symptoms will vary according to the part of the organism chiefly affected by this venous stasis: dysnpœa, cough, cardiac asthma, pulmonary congestion, hemorrhage and œdema of the lungs; cyanosis, œdema of the extremities, ascites, hydrothorax; congestion of the liver, indigestion, occasionally jaundice; congestion of the kidney, with scanty and albuminous urine; headache, restlessness, sleeplessness, irritability, and sometimes more serious nervous and mental disturbances. The physical signs vary with the lesion and with the valve affected and need not be rehearsed here.

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Treatment.—The treatment of chronic endocarditis in the class of patients under consideration would seem to consist primarily in devising some means of lessening their cardiac strain. If the patient is seen early and this end can be obtained, he may reasonably look forward to many years of useful work. The two most important factors in obtaining it are: first, personal instruction in cardiac hygiene, second, the obtaining of some less arduous work than that which has brought about or favored the disease. In our clinic we are constantly urging patients to seek positions such as watchmen, janitors, or messengers, or other such places, where they may earn a livelihood with the least effort. It is often surprising how little intelligent workingmen may know of personal hygiene, and especially of its bearing on their cardiac condition. It is important therefore to give definite instructions as to overwork and rest, strain and worry, clothing, ventilation, bathing, diet, and the care of the bowels. The injurious effect of alcohol should also be clearly explained. As chronic anæmia is frequently a factor in these cases, it should receive appropriate treatment. When the case goes on to some grade of cardiac decompensation, the patient should be confined to bed and is usually better cared for in a hospital. As these patients so often have made an effort to work to the last moment possible, and have thus weakened the myocardium out of all proportion, it would seem, to their endocardial lesion, far better results are obtained and more time is gained by them in the end, by prolonging the rest in bed to the limit; that is, far beyond what the patient himself feels is necessary. The chief difficulty usually comes in persuading the patient that this is desirable. In our clinic we do not hesitate to keep patients with only moderately severe decompensation in bed for 2 months, and have found that the results more than repaid us. After the period of complete rest, systematic exercises are instituted that the patient may return very gradually to the amount of exertion necessary to the avocation decided upon. The medicinal treatment of chronic endocarditis is of greatest importance in the stage of decompensation. Digitalis, its products, and congeners are the drugs par excellence in this condition. To give a full account here of the use and effect of digitalis, its indications and contraindications would lead us too far afield. Suffice it to give a few general principles which to-day guide us in its usage. In the failing heart of chronic endocarditis digitalis is seldom, if ever, contraindicated. It must, however, often be used tentatively and with great caution. We give it in full doses and with the expectation of the most brilliant results in cases of auricular fibrillation, especially if they be associated with mitral disease; and in secondary low blood pressure cases, which have retained a fairly good heart muscle. We give smaller doses and look for a slower and less certain result in aortic cases with a regular pulse or with premature contractions. And finally we use the drug with great caution, beginning with small doses, in all cases complicated by severe myocardial disease or degeneration, especially of the fatty type.

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Arteriosclerosis.—Arteriosclerosis, local or general, is undoubtedly the commonest form of circulatory disease due directly to the influence of occupation. Two of its etiological factors are prevalent among many industrial workers, namely, arterial strain and lead poisoning. The former is brought about by prolonged and heavy muscular exercise, especially that associated with much lifting, or by constantly repeated sudden efforts, which produce a great increase in blood pressure, and ultimately a permanent high tension. This condition may be also brought about by occupations that are accompanied by severe and prolonged mental strain or worry. The manner in which lead acts on the arterial system is still somewhat uncertain. It may act directly as an irritant to the arterial walls, and it certainly raises the blood pressure, probably by its action on the kidneys. Increased blood pressure in itself, if sufficiently long continued, inevitably leads to arteriosclerosis. A third factor which is doubtless very prevalent among these patients, though perhaps no more so than among other classes of the population, is intestinal auto-intoxication, more particularly that caused by the decomposition of certain proteids. We find arteriosclerosis very common among stevedores, iron and steel workers, smiths, butchers, and soap boilers, painters and others exposed to chronic poisoning by the heavy metals; as also among those who are subjected to severe mental strain and worry.

Pathology.—In considering the pathology of this disease it must be remembered that in the aorta and larger branches of the arterial tree there is much elastic tissue and relatively little muscle, while the smaller arteries and those distributed to the various organs contain much smooth muscle and relatively little elastic tissue. This of course favors a greater variation in the flow of blood to the different organs by means of vasoconstriction and vasodilation. Two main types of arteriosclerosis are described: the nodular, affecting chiefly the aorta, and the diffuse, which mainly attacks the smaller vessels. The latter is the type usually caused by chronic arterial hypertension, in the absence of syphilis, gout or other infectious or toxic agents. In this, fibrous overgrowth is found in all three coats of the arteries, followed later by degeneration and often by calcareous deposition, especially in the intima and media. The distribution of diffuse arteriosclerosis is variable. Sometimes all of the arteries are more or less affected; more often only certain groups or systems. And on this fact depends the diverse clinical manifestations of the disease.

Symptomatology.—The symptoms, then, express themselves in groups dependent upon the arteries most affected. Hypertension, though by no means a necessary accompaniment of all cases of arteriosclerosis, is constantly present in those cases brought about by muscular strain and lead poisoning. This means that the main visceral trunks and their branches, usually referred to as the splanchnic area, or the renal arteries are sclerosed, frequently both. The following are the more notable symptoms grouped under the heading of the system affected:

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(*a*) Cardiac: Pain in the cardiac area extending to the shoulders and down the arms, frequently hyperæsthesia in the same area, palpitation and a sense of weight in the precordium; sometimes attacks of angina pectoris. Myocardial fibrosis is the natural sequence in these cases, with all the signs of cardiac insufficiency; dyspnœa, congestion of the kidneys, œdema, ascites, hydrothorax.

(*b*) Cerebral: Headache, often severe and obstinate, attacks of faintness and vertigo, nervousness and insomnia, transient attacks of irrationality, aphasia or partial paralysis. The retinal arteries are often affected early, and give a characteristic picture, accompanied by scotomata and some dimness of vision. Cerebral hemorrhage may be the final outcome.

(*c*) Renal: Chronic interstitial nephritis of a mild grade is very frequently present, and the more severe forms sometimes supervene.

(*d*) Abdominal: Disturbances of digestion are often the earliest symptoms manifested, but unfortunately for purposes of diagnosis are in no way characteristic. Attacks of severe abdominal pain, often seen in lead poisoning, are most likely due to spasm of the partially sclerosed abdominal vessels.

(*e*) Peripheral: A similar spasm of the vessels of the extremities is the cause of the less frequent attacks of intermittent claudication, with severe pain and intermitting loss of function.

Treatment.—All that we can hope to accomplish by the treatment of an existing arteriosclerosis is to arrest its progress, and to relieve some of its symptoms. Should the patient be seen early, when hypertension is alone present, in what has been called the presclerotic stage of arteriosclerosis, much more may be hoped for. In either case, the measures that are most useful are unfortunately often those that it is difficult for the workingman to carry out. Of the first importance is the avoidance of the causes of hypertension, and in most cases this means change of occupation, so as to obtain more rest and greater freedom from strain, or, to avoid the further saturation of the body with lead. Systematic exercise, however, which must be often be insisted upon among the leisure classes, is usually unnecessary in the cases under consideration. The diet is important, as even those engaged in laborious occupations often eat too much, or injudiciously. The proteid intake should be reduced as much as is consistent with the maintenance of strength, and those forms of proteids used that experience has shown are least injurious. My own preference is for cheese and eggs, and with a moderate amount of these together with the proteid obtained from breadstuffs and some of the vegetables and fruits, the 70 to 80 grams a day necessary for a hard-working man may be obtained. If meat is allowed at all, it should be eaten sparingly, not oftener than once a day. Medical opinion varies somewhat as to the deleterious effect of alcohol in arteriosclerosis, but certainly in the forms of the disease under consideration, it is better avoided altogether. The bowels should be kept unusually free, and this can, as a rule, be accomplished in those of an active life by

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careful dieting. If not, laxatives may be necessary from time to time. It is better to resort to them as often as need be, than to allow constipation to persist, with the resultant resorption of a greater amount of poisonous substances. Frequent bathing is desirable, a tepid sponge in the morning or a warm bath at night, and the sweating incidental to labor is a distinct advantage. Too much cannot be expected from medicinal treatment. Potassium iodide is the classical remedy, and if given in a suitable vehicle in small doses, it is usually well borne and seems to be of undoubted service. The nitrites are often valuable in combating the symptoms dependent on hypertension, though they should not be given constantly with the idea of permanently lowering the pressure. What is not accomplished in this way by hygiene, will hardly be by drugs; and indeed it is now a commonplace that the optimum pressure in arteriosclerosis, though often less than what is found in an untreated case is usually well above the normal. Finally, we have to deal with the cases of secondary low blood pressure due to a failing heart. Here digitalis in full doses is indicated, and is often followed by brilliant results.

DISEASES OF THE KIDNEYS

Acute Nephritis.—Acute congestion of the kidneys, followed usually by acute nephritis, as an occupational disease, is caused by the inhalation of the concentrated fumes of turpentine, vanadium, arsenic or carbolic acid, and not infrequently occurs among the workers in these substances.

Pathology.—In these cases the kidneys are usually large, soft, and either red from the large amount of blood present or grayish yellow with fatty degeneration; the capsule strips easily; the tubular epithelium is swollen, hazy, granular and contains fat droplets; the glomeruli are congested and their capsules filled with blood cells and débris. There is at least some inflammatory œdema in the interstitial tissue.

Symptomatology.—The onset is usually sudden, with headache, pain in the back, nausea, vomiting, pallor and the early development of slight œdema. Fever is slight or absent. The urine is scanty and may at first be completely suppressed; the specific gravity is high; it contains much albumen, blood and casts. Acute uræmic symptoms occur in a certain number of cases—vomiting, mania, convulsions or coma. Although always a serious disease, there is a natural tendency to recovery with complete restoration of function after a course of from several weeks to several months.

Treatment.—A minimal amount of work should be put upon the kidneys by reducing metabolism and body waste, and utilizing other methods of elimination. The patient is put to bed on a diet yielding about 50 grams of proteid and 2000 or 2500 calories. This is best obtained by milk and cream, with a small amount of cereal, breadstuffs and sugar. If œdema is present the amount of fluid and of sodium chloride should be greatly restricted. Free catharsis should be maintained. Diuretics may be used, but are of

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doubtful value. If uræmia threatens, sweating and free venesection are indicated. Iron should be given during convalescence.

Chronic Interstitial Nephritis.—This is the more common form of renal disease resulting from occupation, and is met with among workers in lead and other heavy metals, or occurs as an accompaniment of arteriosclerosis from overstrain.

Pathology.—The kidneys are usually small, the capsule thickened and adherent, the epithelium degenerated, the vessels sclerotic and there is a marked overgrowth of the interstitial connective tissue. The heart is always hypertrophied and the arteries usually sclerosed. Anæmia nearly always develops in the course of the disease.

Symptomatology.—The disease is usually insidious in its onset, and is first recognized as a result of a urinary examination, or by the occurrence of a serious complication. Early symptoms are failure of strength, headache, dizziness, sleeplessness, loss of appetite and dyspepsia, and moderate dyspnœa on exertion. The urine is increased in quantity, is of low specific gravity, contains a trace of albumen and a few casts. Disturbances of vision may occur, with retinal hemorrhages or albuminuric retinitis. The pulse is tense and full and the systolic and the diastolic blood pressures increased. The later developments of the case are usually those of chronic uræmic poisoning, nocturnal dyspnœa, with Cheyne-Stokes respiration, uncontrollable vomiting, local palsies, mania, convulsions, coma; or of cardiac decompensation, described above.

Treatment.—Pathologically the disease is incurable, but much may be done to prevent its progress and keep the patient in a fair degree of health. The causes of kidney irritation or of arterial strain should be obviated. This usually means a change of occupation, with the adoption of one that is less strenuous, or in which chronic poisoning from any source may be avoided. The diet should be light and nourishing, similar to that recommended for arteriosclerosis. If active work is to be continued 70 to 80 grams of proteid a day may be allowed; if a sedentary life is adopted, 50 grams is sufficient. The fluid intake should be restricted to about 2 quarts, to relieve the kidneys of extra work. The bowels and skin require careful attention. Sweating and active catharsis may be called for later, with the nitrites, if the early symptoms of chronic uræmia supervene. Iron is nearly always required for the anæmia, and digitalis in full doses for the later cardiac insufficiency tides over many a crisis.

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DIVISION III

Fatigue and Occupation. Neuroses

CHAPTER I

FATIGUE AND OCCUPATION

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Fatigue is a corporeal condition which ought to be taken account of in all human activities, and especially is this true of the activities of industrialism.[1,27] In the now prevalent discussion of industrial efficiency fatigue has remained largely in the background and, whenever it has entered into consideration, what has been written about it has too often revealed an ignorance of physiological knowledge. In its relations to disease, too, the importance of fatigue has never been adequately recognized, and here again industrialism seems to offer many examples of the general principles. Before proceeding to discuss these, it will be well to get in mind a clear picture of the nature of fatigue.[2]

Fatigue in the individual has its subjective and its objective aspects and hence may be considered from the two standpoints of the psychical and the physical. After he has been active mentally or physically the average man "feels" tired; that is, to him fatigue is a matter of sensation, and his sensations are the sole measure of his weariness; he has no comprehension of any material changes in his body which accompany his feelings. Yet important material changes have occurred. Many of them, however, are still unknown even to the specialist in fatigue and, indeed, both the psychical and the physical aspects of the subject are much in need of intensive investigation.

Fatigue in the usual sense of the word may be defined as a diminished capacity for work which is the result of previous work. This final clause imposes upon the use of the word a somewhat too narrow restriction, for diminished capacity for work, indistinguishable from that due to previous work, is often present when previous activity has not occurred. Thus, disease may put the body into such a condition that its working power is lessened; lack of food may do the same; the excessive use of one set of muscles may diminish the power of other muscles or of the brain to work. These cases have certain causative factors in common and it is often convenient to use the word "fatigue" so as to include a considerable variety of

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such instances. Thus a usage of the word sharply limited to a state following work is not desirable.

Fatigue of Tissues.—Diminished capacity for work may be demonstrated very clearly by a single muscle.[2] If the muscle of an animal, such as a frog or a cat, be removed from the body immediately after death, be attached to a weighted recording lever which writes on a slowly revolving drum, and be stimulated by electric shocks at regular intervals, a graphic record may

FIG. 6.—Series of contractions of a frog's sartorius muscle, excised and stimulated at intervals of 2½ seconds. The contractions at first increase in extent, this stage constituting the treppe, reach a maximum, and later decrease, this stage constituting fatigue.

be made of the resulting contractions as a series of vertical lines (Fig. 6). The contractions at first increase in extent, as is indicated by a progressively increased height of the successive lines. This is followed by a period of only slight change in their extent and later by a progressive decrease. These three phases are called respectively the period of the treppe, or staircase, the period of maximum contractions, and the period of fatigue. Such a record is an epitome of the behavior of the working muscle within the intact

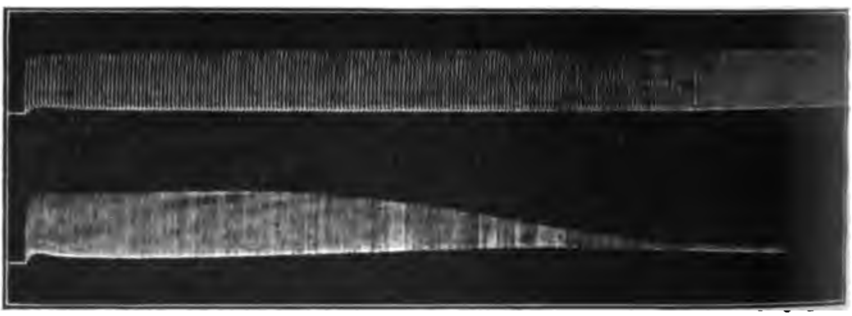

FIG. 7.—Series of contractions of the two excised gastrocnemius muscles of a frog, lifting equal weights, the upper stimulated at the rate of 20 times, the lower at the rate of 45 times, per minute. Note the earlier fatigue of the more rapidly working muscle.

living body, athough in the latter case all the phases of the action are greatly prolonged. If the muscle be stimulated more rapidly (Fig. 7), or be made to lift a heavier weight (Fig. 8), fatigue comes on more rapidly. If at any stage in the fatigue the stimulation be stopped and the muscle be allowed to rest, there is a restoration of working power which is more or less pronounced according to the conditions of the experiment. If a circulation of blood or other appropriate oxygenated liquid be established through the muscle,

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recovery may be complete (Fig. 9). All kinds of muscle are capable of fatigue, not only the skeletal muscles of the limbs and the trunk, but the muscle of the heart and the unstriated muscle of the other viscera. All of the phenomena mentioned have their counterpart within the intact living body, whether of the lower animal or of man.

The diminution in the capacity for work which is so clear in the fatigue of muscle has been demonstrated, although less clearly, by laboratory

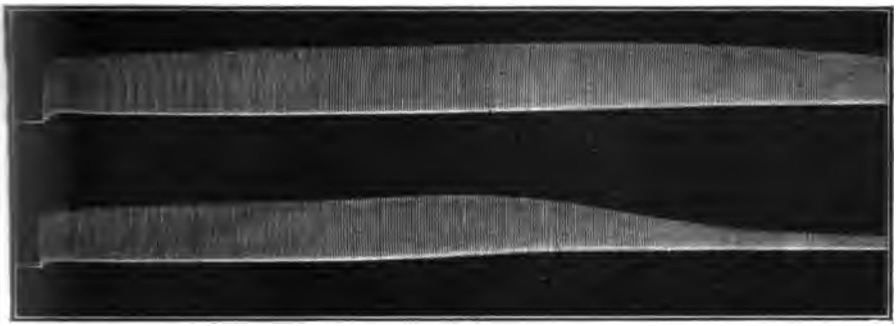

FIG. 8.—Series of contractions of the two excised gastrocnemius muscles of a frog, stimulated at the same rate of 27 times per minute, the upper lifting 10 grams, the lower 30 grams. Note the earlier fatigue of the muscle doing the more work.

experiments on certain other tissues and is undoubtedly characteristic of all living substance. Much effort has been expended in attempting to learn whether nervous tissue, either within or without the central nervous system, is capable of fatigue. This effort was for a long time blocked in the case of nerves themselves, which seem to exhibit no signs whatever of fatigue after

FIG. 9.—Series of contractions of a frog's gastrocnemius muscle *in situ* with the circulation of the blood undisturbed. The muscle was stimulated for 9 minutes, was allowed to rest for 15 minutes during the break in the series, and was then stimulated for 9 minutes. Note the recovery from fatigue during the brief rest.

being stimulated for even many hours. More recently it has become possible to detect a decrease in their electrical current of action and their irritability and thus a diminished capacity for work after nerves have conducted nervous impulses for a considerable period of time. It is not at all certain, however, that this occurs in the intact living body, where nerves seem to be extraordinarily resistant to the deleterious results of previous activity. So within the brain and spinal cord it has been difficult to demonstrate fatigue,[2]

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and the idea is prevalent, also without wholly satisfactory experimental evidence, that while the central nervous system is capable of fatigue no part of the nerve cell, or neurone, is readily fatiguable, and that when nervous fatigue does appear, it is localized primarily in the synapses or semi-permeable membranes at the junctions between successive neurones.

Chemical Changes in Fatigue.—The diminished capacity for work that appears in living substance engaged in activity is a sequence of specific chemical changes that occur in the substance and are a part of its metabolism.[1] These are of two kinds: First, material that is essential to activity is gradually used up and thus the stock of available material becomes gradually exhausted; if this process continues without new material being supplied, activity must in time necessarily cease. Secondly, there appear katabolites, or products of activity; these are poisonous to the living substance and, if allowed to accumulate, gradually diminish its working power; they are therefore often called "fatigue substances." These two processes constitute the chemical causes of fatigue. They occur simultaneously, and in a given instance it is hardly possible to determine their relative importance in the fatigue process.

As to the identity of the chemical substances involved, the evidence is again clearest in the case of muscle. When muscle contracts, its available oxygen enters into chemical combination with other substances, and its glycogen, which is the chief source of muscular energy, undergoes disintegration. Unless oxygen and glycogen are replaced as fast as they are used up, the muscle in time ceases to contract. Carbon dioxide and lactic acid are produced within active muscle and, unless they are at once removed, they react deleteriously on the muscle cells, diminish their irritability, and contribute to their fatigue. When in small quantity, however, carbon dioxide and lactic acid seem to act in the opposite way and temporarily to increase the working power of muscle. To this latter action the author[2] has ascribed the treppe, or preliminary period of progressively increased contractile power, which is present in the early stages of a prolonged period of muscular work. That other metabolic processes in the muscle share in the causation of fatigue is not known but is not improbable. An endeavor[3] has been made experimentally to demonstrate the existence in fatigued muscles of a specific toxin of fatigue, resembling in various properties bacterial toxins, and although it may be true, as claimed, that the extract of fatigued muscles contains material which when injected into animals seems to produce a condition of fatigue, the identity of a specific toxin is not clearly established. The relations between fatigue and metabolism in other tissues than muscle are not yet elucidated. Diminution of oxygen and increase of carbon dioxide can of course diminish the working power of all living tissues. It is conceivable that any katabolic process prolonged without corresponding anabolism may in time diminish the working power of the tissue involved, but the whole subject of metabolism in fatigue is much in need of investigation.

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Fatigue in the Human Body.—When we turn from the fatigue of specific tissues to the phenomena of fatigue in a complex organism like the human body, the problem becomes much more difficult. Here the tissues do not act separately; the excessive work of one increases the work of others. Contracting voluntary muscles must be directed by an active nervous system, and they demand more work by the heart and the respiratory organs and induce more secretion by the various glands. All of these actions in turn involve the nervous system in greater activity. Drain upon a store of metabolic material in one organ is followed by a call for a supply elsewhere in the body. Moreover, if a deleterious katabolic substance is produced in quantity by one organ it passes in circulating blood and lymph throughout the body, and may diminish the working capacity of other organs or tissues. In these ways fatigue of one part of the body may induce the phenomena of fatigue in other parts—local fatigue may result in general fatigue. The exercise of one set of muscles, for example, will fatigue most those muscles and the corresponding parts of the brain and spinal cord, but it will result also in some degree of fatigue in other parts of the body. A person who has worked sufficiently hard to tire his muscles may have a tired brain as well, and the exhausted brain worker is in no condition for performing hard physical labor. A change of work is often beneficial as a temporary expedient to relieve a part of the body that is in danger of becoming exhausted, but there are limits to the possiblity of such benefits, and it ought always to be borne in mind that excessive work by one part means a certain degree of fatigue for all parts.

It is now generally conceded that, contrary to popular belief, sensations of fatigue are no measure of diminished capacity for work. It has been demonstrated by careful laboratory tests that a given task may be performed as effectively and as readily whether the individual feels fresh or tired. Thus, a student, although feeling used up and incapable of further work, may be able to solve arithmetical problems just as quickly and correctly at the end of a series of such tasks extending over many hours as at the beginning. A feeling of fatigue often may induce a disinclination to work and even a powerful aversion toward it, and this may diminish the amount of work accomplished, although at the same time not decreasing capacity. A slight change in one's mental state often makes a great difference in achievement. Soldiers can march farther and with less feeling of fatigue when music accompanies them. Ayres[4] found that in a 6-day bicycle race the average number of miles ridden per hour on one of the days of observation was 18.8 with no music, and 21 when the band played.

Recovery from Fatigue.—Comparatively little is known of the details of the process of recovery from fatigue. Adrenin, the internal secretion of the adrenal bodies, when administered to a fatigued muscle appears to have a powerful effect in increasing its irritability and its contractile power.[5] Whether it does this by a direct action on the muscle substance or by neu-

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tralizing or destroying the toxic fatigue substances is not clear. Nor is it clear whether it acts constantly or only in emergencies. Some investigators[5] have claimed the discovery of a specific antitoxin of fatigue, but the existence of such a body is doubtful. It is obvious that rest brings recuperation. During rest there is an opportunity for the excess of carbon dioxide, lactic acid, and whatever other fatigue substances may be present, to be eliminated from the fatigued tissues and from the body. The carbon dioxide is passed out of the body through the lungs, while the other fatigue substances probably either pass out unchanged in the urine or are broken down into simpler substances before being discharged. At the same time rest gives an opportunity for the rebuilding within the cells of the substances that are necessary to further activity.

Tests for Fatigue.—Much ingenuity and time have been expended by investigators in efforts to discover reliable objective signs of fatigue in human beings and to devise ready methods of demonstrating them. Many of

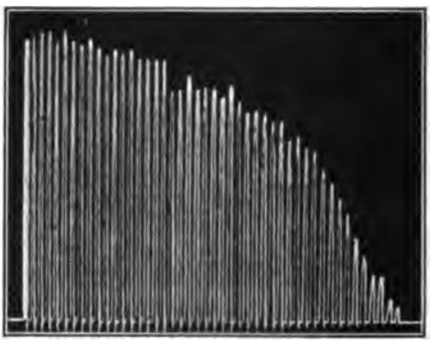

FIG. 10.—Series of voluntary contractions of the flexor muscles of the middle finger lifting a weight of 3 kilograms. Note the progressive fatigue. (*From Mosso.*)

these efforts have failed. One of the earliest contributions was that of the ergograph of Mosso[6] which records the contractions of a certain set of muscles, such as those involved in the bending of a finger, when stimulated at regular intervals, either by the will through the nervous system or by electric shocks directly applied to the muscles, and when made to lift a given weight (Fig. 10). The ergograph was believed to afford an exact method, easily applied, by which bodily fatigue can be readily demonstrated, and it was extensively used to obtain data for the formulation of the laws of human fatigue. It has now been shown, however, that ergographic records are really significant only when made by a trained subject. Even then the fall in the curve as the work progresses is no real measure of ultimate fatigue of either the muscles or the nerve tissues. It is doubtful whether it reveals more than a temporary lack of oxygen in the neuromuscular mechanism. A slight diminution in the weight to be lifted often shows that the mechanism is in good working condition and is capable of working for an indefinite period.

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Various so-called mental tests, such as speed and accuracy in the performance of arithmetical problems, the speed and accuracy of perception and of certain movements, attention tests, memory tests, the estimate of time, the degree of sensitivity of the skin to stimuli variously applied, and others, have all contributed valuable data,[7] yet they require intelligent trained subjects under exact experimental conditions and the expenditure of considerable time, and even when all the requirements for accurate observation are provided they cannot be relied on to reveal unerring signs of fatigue.

The production of fatigue substances, except carbon dioxide, while undoubted, cannot be readily demonstrated in the living man by our present methods. It is claimed that in the urine of fatigue there is less nitrogen combined in other substances than in urea. Although the alkalinity of the blood and the amount of sugar present in it are said to be decreased in fatigue, such chemical tests are hardly practicable in the human body as routine procedures. No direct relation between bodily temperature and fatigue has been observed. The distribution of the blood in the body is probably altered by a long-continued fatiguing task: by the end of a hard day's work, either mental or physical, much blood has probably been drained away into the limbs and the brain is left relatively anæmic, this doubtless accounting in part for the feeling of sleepiness that is then present. Preliminary experiments by the author and others seem to make it possible that a careful study and evaluation of vascular conditions, such as blood pressure and heart rate, may reveal reliable objective signs of the presence of fatigue. But until adequate and readily applied methods of quantitative measurement and accurate localization in the intact individual can be devised, fatigue as a topic of exact science must remain in an unsatisfactory state. The lack of such methods is especially regrettable in the field of industrial occupations. Here our conclusions as to the presence and the degree of fatigue must for the present be largely a matter of indirect inference rather than of direct demonstration.[27]

Fatigue and Disease.—The connection between fatigue and disease is probably much more frequent and close than is usually recognized. To disease fatigue stands in the relation of both cause and effect. That it may be a factor in causing disease is, indeed, often believed, and this belief is justified by laboratory experiments. Thus, it has been shown experimentally that of two groups of animals, the one resting and the other fatigued by muscular work and both inoculated by pathogenic bacteria, the fatigued animals exhibit a fall of the opsonic index and succumb to the disease in larger numbers.[8] This may best be interpreted as indicating that the resistance of the body to the action of bacteria is diminished by the metabolic changes involved in the fatigue processes. If this is true of one species of bacterium it is probably true of others, and from the prevalence of the infectious diseases in the life of man it is obvious how important is the matter of avoiding undue fatigue. Fisher[9] has written graphically: "The

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typical succession of events is first fatigue, then colds, then tuberculosis, then death. Prevention, to be effective, must begin at the beginning."

Outside the realm of infections, fatigue as a causative factor in disease is perhaps most obvious in neurasthenia. The most common single precedent of this form of neurosis is excessive activity of the nervous system. Continued muscular fatigue, if it is without accompanying excessive excitation of the nervous system, probably cannot induce neurasthenia; the nervous system is the primary seat of the preceding fatigue process. There are, of course, many contributing causes, but overwork, overpressure, overstrain are the unavoidable precedent of the nervous breakdown.

The immediate causative relation of fatigue to other diseases than those mentioned is neither so obvious nor so clearly established, but it is probable. Fatigue in a normal degree is a harmless, even a healthful, phenomenon—in its essentials metabolic—which involves a physiological depression of the bodily functions and induces its own termination and a return to the former capacity for work, but it can easily be carried too far; destruction of cellular material and the accumulation of toxic katabolic substances unwisely persisted in without adequate opportunities for recuperation can proceed to a pathological degree and interfere profoundly with the normal metabolic processes of the organism. Thus the foundation of profound diseased conditions may be laid.

That fatigue, or at least proneness to fatigue, is an effect of disease is too obvious to require discussion; diminished capacity for work is the one almost universal accompaniment of diseases that involve alterations of general metabolism, such as infections, febrile diseases, and diabetes. Here we find the two general metabolic processes that characterize fatigue even more prominent than in normal states. The loss of substances that are essential to activity is well illustrated by the destructive metabolism of febrile diseases and the excretion of sugar in diabetes mellitus. To the normal fatigue substances, which may or may not be present in disease in abnormal amounts, there must here be added a considerable number of other toxic substances which from their depressing action may not inappropriately be called "pathological fatigue substances." This group includes probably the whole range of bacterial toxins and a considerable number of intermediate metabolic products derived from the body's own cells. Whether in perverted metabolism intermediate metabolic products that are new to the organism are formed, or whether the change consists in an increase in quantity of normal metabolites, are questions not yet settled and need not be discussed here. Whatever their origin, examples of pathological fatigue substances are oxybutyric acid, which is common in diabetes mellitus, and indol, which is common in intestinal putrefaction. These substances are known to be depressing to living tissues,[10] and whenever they are present in quantity in the living body, fatigue is a prominent symptom. Further research will, without doubt, reveal the depressant action of other products of pathological

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metabolism, especially the acids occurring in acid intoxications. Here is a wide field for research. Research is much needed also in such conditions as neurasthenia, myasthenia gravis, and various other diseases which are characterized by excessive susceptibility to fatigue. In these conditions it is justly conceivable, but not proved, that faulty metabolism, resulting in the occurrence of pathological fatigue substances, is responsible for the characteristic symptoms.

Fatigue and Industrial Output.—In modern industry fatigue plays a rôle of prominence and is capable of demonstration in various ways. In the first place, it diminishes the output of the worker. An Italian physiologist, Pieraccini,[11] has made a study of the amount of work performed at different hours of the day by several manual workers, such as type-setters, stone-

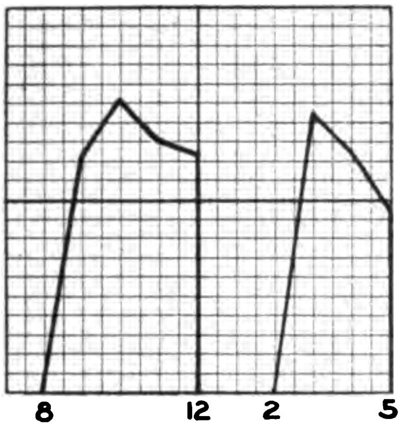

FIG. 11.—The distribution during the working day of the total output of six typesetters working at piece rates. The number of lines set during each hour was as follows: 8–9, 121; 9–10, 151; 10–11, 130; 11–12, 125; 12–2, rest and lunch; 2–3, 142; 3–4 124; 4–5, 96. (*After Pieraccini.*)

cutters, nail-makers, diggers and bullet-makers, and has found that it increases rapidly during the early part of the forenoon, declines toward the noon hour, increases again to a lesser maximum after luncheon, and then rapidly falls off to a low minimum at the end of the day's work (Fig. 11). The total output of the afternoon is always less than that of the forenoon, and the quality of the afternoon work, as measured by the number of errors, is poorer. From the study of a variety of industries the Committee of the British Association[1] summarizes the output of the successive 5 hours of a spell of work as "small, very great, great, fair, small." Roth[12] reports that in a large rolling and steel mill 57.5 per cent. of the output was produced during the first half and 42.5 per cent. during the second half of the shift. This general result has its counterpart in the paradox that diminishing the hours of labor increases the total output, a fact that has been demonstrated often enough to be accepted as true within reasonable limits. Industrial

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fatigue is also shown in an inferior output toward the end of a long working day and even on the succeeding day, facts that are abundantly attested by experience.

Fatigue and Health of Industrial Workers.—One of the most striking evidences of the existence of fatigue and its seriousness in industrial work is found in a consideration of the health of workers. Here we must distinguish between localized and general affections. Localized affections comprise those in which one part of the body, such as a group of muscles with their nervous connections, is overstrained by long-continued use. Here are to be grouped the fatigue neuroses, such as writers' cramp, telegraphers' cramp, and the various other spasmodic muscular conditions that occur in violin and piano players, seamstresses, shoemakers, hammermen, and others, as well as miners' nystagmus and the professional torticollis of tailors and cobblers. The seat of these pathological conditions is probably the central nervous system; they have, however, not yet been critically analyzed sufficiently from the physiological standpoint, and their real nature is therefore unknown. The ascription to fatigue as the causative agent of the more general diseases from which industrial workers suffer is not so clear as with the localized affections. It is doubtful, for example, whether the proneness of locomotive engineers to diabetes is rightly to be attributed, as has been done, to the nervous strain of their work. With nervous disorders and especially neurasthenia, however, the connection seems quite evident. There can be no question that this state of chronic generalized fatigue, which has usually been supposed to be peculiar to professional men, men of large affairs and the wealthy unemployed, is often directly the sequel of the cumulative fatigue of the long-continued, monotonous, wearing labor of industrial workers. There is a general consensus of opinion among those best fitted to know that in recent years there has been a marked increase of neurasthenia among working people, and that this is due largely to the overstrain of their occupations. Leubuscher and Bibrowicz[13] showed that at the large industrial sanitoria at Gütergotz and Beelitz in Germany 26 per cent. of the patients were neurasthenics, and the percentage of such cases for individual years rose from 18 in 1897 to about 40 in 1904 and 1905. Some of these cases were classified according to occupation as follows:

Type-setters	15.75 per cent.
Carpenters	9.45 per cent.
Locksmiths	5.00 per cent.
Mechanics	1.90 per cent.

In a study of 200 cases of neurasthenia and hysteria in the Industrial Sanatorium at Zehlendorf, Schönhals[14] found the distribution between skilled and unskilled workers to be as follows:

Artisans, highly	skilled, 57 per cent.		74 per cent.
General workers	skilled, 17 per cent.		
	unskilled, 26 per cent.		

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A marked percentage of these workers were engaged in factories, where the strain of work is apt to be great. Cardiac disorders of nervous origin are common among working-people. Roth[12] has pointed out the prevalence of anæmia among young women workers, especially those in textile factories, shop-girls, sewing women, and maids in hotels and public houses, among all of whom overwork is common.

Fatigue and Borderland of Illness.—Even where diminution of output is not present and where specific diseases cannot be traced directly to the fatigue of labor it is undoubted that industrial overwork often occurs and puts the worker into a physical condition, at present difficult to recognize by any specific test, wherein his physiological mechanism is in a state of depression and ready to fall a prey to specific maladies. Treves[15] speaks of this as not presenting "a well-defined morbid picture; but it is a slow deviation, often obscured by its very slowness, and predisposing to illness of any nature; it is the borderland of illness." The future careful study of individuals will doubtless make this condition more precise.

Characteristics of Modern Industry.—It is a common belief that fatigue and its resulting evils are relatively more common among industrial workers in recent than in earlier times. Notwithstanding the many evils of the earlier human slavery, now happily nearly extinct throughout the world, this belief is probably in the main correct. Greater fatigue is to be expected from the specific characteristics of modern industry. While these differ in detail in different industries they frequently include the following: The great division of labor and the consequent specialization of the work of each individual, the speed of the work, certain other factors of the work, the imposition of long working periods and of overtime work, the introduction of piecework, and the crowding together of large numbers of human beings within confined spaces often poorly ventilated. These will now be considered in detail.

Specialization and Speed of Work.[1, 16]—A pronounced feature of modern industrialism is the great division of labor among the workers and the limitation of the task of each to a specific procedure. While certain kinds of work still require the expenditure of much muscular force by the worker, the introduction of machinery has tended in general to diminish muscular effort. It has, however, been replaced by a new element which is no less fatiguing, namely, speed. Thus, in the making of hinges a woman lifts a half-formed hinge, places it in the bending machine and quickly withdraws her hand, and repeats this series of movements at the rate of 50 times a minute, or 30,000 times a day. The tops of tin cans are cut by pressing the lever of a foot press 40 times a minute, 24,000 times a day. In the telephone service an operator can receive, answer and make the proper connections for from 200 to 300 calls in an hour; in weaving one woman must supervise 16 to 24 looms, ever watchful that they are running properly; in sewing a single girl watches intently the 12 jumping needles of her power

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machine; in the making of women's clothing by modern machinery one operator in an hour will tuck 250 yards of lawn, another will hem 400 yards of voile, another will make 1000 buttonholes, and still another will sew on 800 buttons; in the manufacture of candy one employee will wrap 9000 caramels in a day; and in a cigar factory one man will bunch 2000 stogies. An expert can insert in one day the eyelets into 4000 shoes; another can trim the superfluous leather from the uppers of 5200 shoes. A machine-made shoe in the process of manufacture is said to pass through the hands of no less than 100 workers. A worker doing one thing does nothing else, that is, his main activities are limited to a small part of his body, to a restricted neuromuscular mechanism, which undergoes a rapid rhythmic exercise. In some cases this exercise becomes hardly more than a series of exactly similar unconscious reflex actions; in others it demands the aid of an acutely attentive consciousness. The danger lies in the pace becoming so rapid that there is little opportunity, such as usually exists with the rhythmically beating heart, for recuperation between successive discharges of energy. At the end of the day's work, therefore, the physiological mechanism involved is too often near exhaustion and even the rest of the body may suffer likewise.

We have here, indeed, a condition strikingly like that of the single excised muscle of the familiar laboratory experiment (Fig. 6): With the excised muscle the stimuli are electric shocks regularly and rapidly repeated; with the industrial worker there is a restricted group of muscles stimulated rhythmically from a particular part of the nervous system. In both cases necessary metabolic material is consumed and fatigue substances are produced. With the excised bloodless muscle there is no replacement of the one or the removal of the other, and the stage of exhaustion is quickly reached; with the human muscles and the associated nervous tissues fuel and oxygen are brought and wastes are removed by the blood, but with the great speed of stimulation katabolism is pretty sure to exceed anabolism, and thus favorable conditions are provided for the production of pronounced fatigue in the parts involved and a lesser degree of fatigue in other parts of the body. The lesson of Fig. 7 is here directly applicable.

Certain Other Factors of Work.—Besides speed there are certain other features of industrial work which contribute to fatigue. This is obvious, for example, in the lifting of heavy weights. Fig. 8 shows clearly that the heavier the load the earlier is the muscle exhausted. Processes that involve the jarring of the body are probably also similarly detrimental. This is a feature of the action of certain kinds of heavy machinery and especially of pneumatic tools. The excessive noise of some kinds of machinery, especially looms, acts to distract the attention and thus makes more wearing the specific task of the worker. This is doubtless a greater factor in the production of fatigue when the noise is variable in intensity and uncertain in its onset. It is conceivable that a worker may become accustomed to

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and learn to neglect a constant hum or roar in his environment. Faulty or strained postures of the body, necessitated by the holding of tools in certain positions or the tending of machines, are a fruitful and often unnecessary source of bodily fatigue.

Length of Working Period.—The tendency of modern industrialism, actuated by commercial motives, has been to demand of laborers the longest possible working period. More than a century ago humanitarianism, urged by the evils of excessive labor, began to protest against this unbridled overtaxing of human beings and undertook to secure legal limits to the length of the working day. The passage by the British Parliament in 1802 of the Health and Morals of Apprentices Act marks the beginning of such legislation. The science of physiology can now provide even stronger arguments for reducing the hours of labor than can humanitarianism, and in recent years there is here a tendency to utilize physiological knowledge. That there is still great diversity of custom as to duration of labor in different countries and in different trades is shown by the following tables:

RELATIVE PERCENTAGE OF WEEKLY HOURS OF LABOR IN DIFFERENT COUNTRIES, THOSE OF THE UNITED STATES BEING TAKEN AS 100[17]

United States	100
England and Wales	104
Germany	115
France	121
Belgium	126

WEEKLY HOURS OF LABOR AND PERCENTAGE OF LABORERS IN INDUSTRIES IN THE UNITED STATES EMPLOYING 25,000 OR MORE PERSONS[18]

	48 hours and under	Between 48 and 54 hours	54 hours	Between 54 and 60 hours	60 hours	Between 60 and 72 hours	72 hours and over
Cotton	0.1	0.8	0.1	50.4	31.5	17.0	0.1
Hosiery and knit goods	0.7	2.6	3.7	50.6	38.6	3.8	....
Woolen goods	0.2	0.6	0.7	71.0	26.8	0.6	*
Silk	0.1	2.3	2.3	82.2	13.0	....	....
Cordage and twine	0.5	0.8	9.3	63.0	23.3	2.6	....
Dyeing and twisting textiles	0.2	2.5	0.9	64.0	28.7	3.6	....
Iron and steel blast furnaces	....	....	....	0.5	3.0	10.6	86.0
Steel works and rolling mills	7.6	1.7	10.0	12.1	34.2	12.6	21.8
Electric machinery	1.3	16.0	24.0	52.2	6.7	....	*
Shipbuilding	9.5	6.6	24.4	24.8	34.7	*	....
Agricultural implements	0.9	4.0	8.1	54.5	32.3	0.3	*
Slaughtering	5.5	4.6	11.7	4.8	72.2	0.7	0.5
Flour and grist mills	8.6	1.5	6.6	4.2	48.3	11.8	18.9
Canning and preserving	4.4	2.2	5.1	7.7	71.6	4.8	4.2
Lumber	3.4	2.5	6.0	7.2	67.5	13.0	0.3
Musical instruments	3.3	6.7	45.7	15.9	28.2	....	....

* Negligible.

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WEEKLY HOURS OF LABOR AND PERCENTAGE OF LABORERS IN INDUSTRIES IN THE UNITED STATES EMPLOYING 25,000 OR MORE PERSONS.—(*Continued*)

	48 hours and under	Between 48 and 54 hours	54 hours	Between 54 and 60 hours	60 hours	Between 60 and 72 hours	72 hours and over
Coke	6.8	1.2	27.2	0.5	39.7	13.3	11.2
Gas	1.3	*	3.0	7.3	15.6	15.5	57.6
Steam laundries	8.4	15.8	19.0	22.4	33.7	0.5	0.1
Turpentine and rosin	37.4	6.7	2.6	0.7	49.8	2.5	0.4
Boot and shoe	0.4	3.3	24.0	57.3	14.7	0.1	*
Leather	0.9	2.8	7.0	39.7	49.2	*	*
Paper and wood pulp	7.4	8.0	4.5	8.7	30.2	19.6	21.7
Printing and publishing	53.7	16.0	18.3	7.4	4.2	0.2	0.1
Automobile	0.4	3.0	30.0	35.2	29.4	*	1.8
Carriage and wagon	4.6	7.0	16.7	31.4	39.7	0.5	*
Glass	16.5	24.1	12.6	20.2	15.6	4.9	5.9
Brick and tile	10.4	2.0	12.2	6.0	66.1	2.6	0.6
Pottery, etc	11.6	10.2	17.9	18.9	39.1	1.7	0.8
Carpet and rug	0.5	0.5	21.3	41.4	36.2	....	....

* Negligible.

In considering the above table it should be borne in mind that 48 hours of weekly labor mean customarily a working day of 8 hours for 6 days in the week and a day of rest on Sunday; 54 hours mean a working day of 9 hours for 6 days in the week, 60 hours one of 10 hours for 6 days, and 72 hours one of 12 hours for 6 days. These are hours of actual labor and do not usually include the period of luncheon, except where the 72-hour week prevails.

The most striking instance among well-organized modern industries of a long period of compulsory labor is afforded by the iron and steel industry of the United States. In 1910 the Department of Commerce and Labor[19] made a very full investigation of the conditions of labor in iron and steel factories and found that of the 172,671 employees of blast furnaces, steel works, and rolling mills, covered by the report, 62.98 per cent. customarily worked more than 60 hours per week; 42.58 per cent. worked 72 hours or more per week; while 20.59 per cent. worked 84 hours or more. A weekly labor of 72 hours means 12 hours every day except Sunday, while one of 84 hours means 12 hours daily including Sunday. The luncheon period is here included, but it is usually brief and desultory. The customary working time of 29.28 per cent. of the employees was 7 days. Once in every week or every 2 weeks, when a night shift was changed to day work and *vice versa*, one shift remained at work continuously for either 18 or 24 hours. Since the above investigation there has been a marked diminution of the proportion of employees who are required to labor every day in the week, but the requirement of 72 hours for a large number of the workers still holds.

In determining the proper length of the working period the factor that

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should be considered first is not the commercial but the physiological one. No one, except in rare emergencies, should be forced by his employer to labor continuously for such a period that subsequent time allowed for rest is insufficient to enable him to recuperate before the beginning of the next working period. Physiological facts, though exact data are here meager, leave no doubt about the necessity, as a custom for most individuals, of one day's rest or change of occupation in every 7 days if one's body is to be kept in an efficient physiological state. The proper length of the working period of the other 6 days ought to depend, first, upon the degree of fatigue that is induced by the specific labor. Here again exact studies are much needed. Different varieties of labor undoubtedly differ greatly as to their fatiguing power and this can be modified by many things, but too little is known of this. Different individuals, too, differ greatly in susceptibility to fatigue. Moreover, the sociological question is a legitimate one, namely: What proportion of an individual's working hours ought equitably to be spent in his vocational work? A customary working day of 12 hours can hardly be justified on either physiological or sociological grounds. The limit in most occupations has, therefore, been gradually reduced, and the present goal of most employees is that of an 8-hour day and a 44-hour week, which signifies a half-holiday on Saturday. In those industries, such as the manufacture of certain iron and steel products, where it is claimed that continuous operation of a plant is necessitated by the nature of the processes involved, either a 12-hour or an 8-hour shift would seem, if the claim is true, to be essential. In the steel industry of Great Britain the shorter working period is gradually being introduced. In the United States movement in the direction of an 8-hour day has been hastened by the adoption of such a day with a 44-hour week for all employees of the national government.

Where there has been an intelligent comparison of the 8-hour and a longer working period in actual experience, the result has proved satisfactory and advantageous to both employees and employers. Thus, the Salford Iron Works, engaged in the manufacture of machinery, at Manchester, England, in 1893 reduced the hours of weekly labor of its employees from 53 to 48, *i.e.*, from a 9-hour to an 8-hour day, and made a careful study of the result. It was found that production, instead of falling off, actually increased. The recent reduction in the steel works of South Wales from a 12-hour to an 8-hour shift has resulted, so far as the figures have been collated, in an average increase of output of 12½ per cent. in the smelting shops and 22½ per cent. in the bar mills. A change from a 9-hour to an 8-hour day was made in 1900 by the Zeiss Optical Works in Jena,[20] also with an increased output in the lessened time. As the workers there were pieceworkers, there were also increased earnings on their part. This result was due, not to a better feeling on the part of the workers, but to the increased speed and effort which they unconsciously exerted. This resulted, of course, in increased fatigue from the actual work of production, but, on the other hand, the shorter

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working period insured two advantages, namely: lessened "passive" fatigue, as Abbe called it, that is, the fatigue of constant attention, fixed attitude, and exposure to noise and confusion; and a longer period for recuperation. Abbe believed that for three-fourths of the industrial workers of Germany 8 hours was sufficiently long to reach their maximum of daily production.

The fatigue of the regular working period is added to by the not infrequent practice of requiring employees to work overtime in periods of emergency. In the canning industry, for example, the demands of perishable fruit or vegetables often induce employers to call back their staff for long evenings of work after the usual day of labor has ended. The Christmas "rush" season in shops is a time of overfatigue for many salesmen. Overtime work is always apt to be strenuous, and the fatigue that results from it is probably relatively less easily compensated for than is that of the usual day's labor.

Whether the universal adoption of a rigid 8-hour work day for all industries would ultimately be best for society and the individual is doubtful. In the ideal scheme of labor it would seem that no person ought to be denied the pursuit of his vocation for a longer period than 8 hours in 24, provided the longer labor is not detrimental to him or to society. The leaders in the world's progress have not limited themselves to brief working days; almost invariably they have been persistent hard laborers with whom, impelled by whatever motive, the accomplishment of the task and not the avoidance of fatigue has been the aim. With the masses who follow instead of lead some limitation is necessary for protection, and an 8-hour day is for the present probably the most just, both physiologically and sociologically. It seems reasonable to believe, however, that when our knowledge of experimental methods has advanced sufficiently, by a careful study of the physiological powers of individuals, on the one hand, and the fatiguing capacities of occupations, on the other, we may arrive ultimately at an adaptation of worker to work which will approach the physiological ideal. This will probably require a work day of variable length, variable both for individuals and for occupations. However this may be, it is obvious that society has not yet found a satisfactory solution of this complex problem.

Piecework.—It might seem that industrial fatigue imposed by long hours might be obviated by paying workers, not according to the time spent, but according to the amount of work accomplished, and, indeed, the piecework system has now become common in many trades. From the standpoint of its theory this system is to be commended, for instead of rewarding all workers alike, whatever their grades of efficiency, it allows those who are ambitious and capable to reap the benefits of their greater powers of accomplishment. But in practice it has developed abuses, for when the rapid worker becomes in the opinion of the unprincipled employer too rapid, piece wages are lowered and further speeding-up is thus demanded. Moreover, the rapid worker is often called upon to set the pace for those who are physiologically slower, and thus they may be urged on at a dangerous rate. The piecework system,

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as thus practised, has become one of the frequent factors in the production of excessive industrial fatigue and has been widely condemned. Its evils are most potent when it is combined with long hours.

Ventilation.—The evil effects of the crowding of many human beings into confined spaces do not come from the chemical vitiation of the air, for the consequent diminution of oxygen and increase of carbon dioxide are not sufficient in amount to produce evil effects, and the hypothetical volatile organic poison of expired air does not exist. These effects, it has now been demonstrated,[21] are due to the increase in the temperature and the humidity of the air, aided by its lack of motion. Increase in surrounding temperature makes it more difficult for the body to throw off by radiation and conduction the excess of heat which it is constantly producing; increase in humidity adds to this difficulty by diminishing the cooling evaporation of perspiration from the surface of the body; while if the air be not in motion the hot humid envelope next the skin does not give place to a cooler dryer layer with its relieving quality. The result is an elevation of bodily temperature, a diminution of vasomotor tone, a gorging of the skin with blood and its consequent removal from the brain and elsewhere, increased perspiration, and the bodily discomfort, sleepiness, headache, and other characteristic sensations of a "close" room. In such an environment there are sensations of fatigue and less inclination to do either physical or mental work, and under extreme conditions actual inability to acccomplish as much with the early oncoming of exhaustion.[28] The following table gives the temperatures in degrees Fahrenheit that have been observed in various work places:—

Starching and ironing in laundries	95
Tending electric furnaces	100–120
Vulcanizing and japanning	90
Evaporating rooms of sugar refineries	110–115
Copper reduction	100
Manufacture of oxygen	100–120
Bakeries	90

Fatigue and Accidents.—Accidents to workmen are caused by something going wrong with either the machine or the man. The larger number of industrial accidents are probably due to the latter. Of 2678 accidents in the state of Illinois in 1910 Bogardus[22] found 17.2 per cent. to result from events beyond the control of the injured—the breaking of machinery, the bursting of boilers, flying pieces of metal—and the remaining 82.8 per cent. to come from lack of proper movements by the workman himself. It is now generally recognized that fatigue is an important factor in the causation of industrial accidents. This is indicated by the time of their occurrence. Bogardus found them to increase in number gradually throughout the forenoon reaching a maximum between 11 and 12 o'clock, to diminish sharply during the hour when most workers are having their luncheon, and to rise progressively to a second and equally large maximum between 4 and 5 o'clock

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(Fig. 12). Summarizing, conversely, the statistics from a large number of industries, the Committee of the British Association[2] states the accident immunity in the successive 5 hours of a spell of work to be "very great, great, fair, small, fair." A progressive increase through both forenoon and afternoon is shown by the statistics of most countries where accurate records have been made, but the two maxima usually occur before the final hour. This falling-off in number toward the end of each half-day is usually ascribed partly to the decrease in the number of persons working, partly to a more leisurely attitude of most of the workers, and partly to a spurt on the part of a few who are stimulated to more acute attention and greater care by the prospective release from work. Besides the daily course of accidents there is in certain industries a weekly progression, more injuries occurring late in the week than early. Monday usually breaks into the regularity of the series by exhibiting a considerable number, which has been ascribed to dissi-

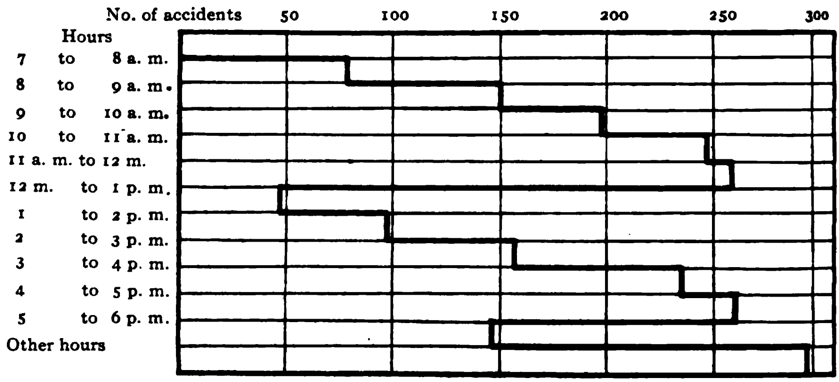

FIG. 12.—The distribution of industrial accidents during the working day. (*From Bogardus.*)

pation on the day of rest; this inference is probably not wholly justified. The day after any holiday is similarly marked.

That fatigue is one of the pronounced causes of industrial accidents can no longer be doubted. Modern industrial work demands on the worker's part keenness of attention and precision of movement. A hair's breadth often marks the dividing line between safety and harm. Without a sharp focus of attention, without accurate coördination of the muscles involved in an act, the workman may come into contact with the molten metal, or touch the moving knife, or become caught in the revolving gear. Both keenness of attention and precision of movement are impaired in fatigue, and thus the work leads the worker on to his own injury. This is often ascribed to his carelessness, and yet in many cases carelessness has a physiological basis in fatigue, and thus we get back to the depression of the physiological powers as one of the physical essentials of liability to accident.

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Scientific Management and Fatigue.—In recent years there has arisen a new system of organizing and conducting industrial work, known as scientific management, and it has been enthusiastically advocated and widely adopted.[23] It aims to find the worker best fitted for a particular task and then to bring him to his maximum efficiency therein. This is done through a careful preliminary study by the efficiency engineer of the task itself, its requirements and the best ways of satisfying them, through the improvement of the machines required, the elimination of unnecessary motions by the worker, the introduction of frequent rest periods, and, in general, the arrangement of all conditions so as to secure the doing of the task in question and no other, and its performance in the quickest possible time of which the worker without detriment to his physiological powers is capable. By eliminating useless motions and concentrating on those activities that upon analysis are found to be essential to the performance of the task much unnecessary fatigue can be avoided. It is obvious, however, that the greater exercise of the particular neuromuscular mechanisms that are involved leads to their greater fatigue—we seem to have here the proper conditions for speeding-up with all its possibilities of overstrain and exhaustion. Anything, however, that produces overstrain and exhaustion diminishes the maximum efficiency of the worker and thus defeats one of the fundamental conditions of scientific management, and hence adequate resting periods, brief but frequent, must be provided. Taylor,[24] the leader in the new system, found that in the handling of pig iron, each pig weighing 92 lbs., a first-class workman should be free from his load during 57 per cent. of his daily working time. With lighter loads this time may be decreased—with a half pig to 42 per cent. of the day. By scientifically systematizing the work of handling pig iron and selecting and educating the workmen, Taylor increased the daily output of each man from 12½ to 47½ tons, apparently without overstraining him. Similarly Gantt[25] by scientific management was able to increase the production in the various operations of a bleachery by 55 to 275 per cent., in small automatic screw-machine work by 25 to 256 per cent., and in large automatic machine work by 30 to 215 per cent. Gilbreth[26] reports that in building a brick wall, while the customary rate was 120 bricks laid by each man in one hour, one who was skilled in the new method, by which the muscular motions required for each brick were reduced from eighteen to five, was able to lay 350 bricks in the same time. In inspecting and selecting bicycle balls 35 girls under the new system were able to do the work formerly done by 120. In all cases wages are materially increased and thus there is added a powerful incentive to the accomplishment of quick work.

In theory scientific management seems to offer a method by which excessive industrial fatigue may be avoided, and in practice at its best it doubtless frequently accomplishes this. Just as, however, the actualities of practice are often far removed from the ideals of theory, so here the great

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danger would seem to lie in the misuse of the new system, in an increase of work done without adequate regard to the powers and the welfare of the worker. Whether such a result actually occurs and, if so, how frequently, we have at present no adequate knowledge. There is little in the literature of the new system to indicate that its enthusiastic apostles understand the nature of fatigue and its manifestations. Its writers, indeed, say little of this phase of their subject. It is obvious that there is much needed the collection of data from the new experience and the comparison of them with the facts of traditional industrialism. Only thus can we learn whether scientific management offers to the worker real release from the unwarranted strain of his occupation.

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CHAPTER II

OCCUPATION NEUROSES

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Synonyms.—Copodyskinesia; dyskinesia; craft neurosis; occupational dyskinesia; Dyskinésie fonctionelle (functional dyskinesia); professional ataxia; (coördinatorische) Beschäftigungsneurosen; affection nerveuse professionnelle; functional spasm; crampe Fonctionelle; crampe professionelle fonctionelle; fatigue disease; anapeiratic paralysis; local chorea; partial vertigo.

The typical occupation neurosis is writers' cramp, for which the variety of names is significant; *e.g.*, scriveners' palsy, crampe des écrivains, Schreibkrampf, steel pen palsy, mogigraphia, emotional dysgraphia, graphospasm, paralysis notariorum, stammering on paper, impotence fonctionelle des écrivains, loss of grip.

Fundamental and Systematic Studies

The disease was apparently first described in modern times by Sir Charles Bell, in 1830, and by a German author, Brück, in 1831. Considerable discussion followed in Germany and France, and was described in Canstatt's text-book of medicine. The first to describe the disease upon modern lines was Duchenne, who developed the theory of disturbed coördination (see below). Benedikt in 1874 added important aspects of a theoretical nature (cell group coördinations in the central nervous system). The first edition of Allbutt's *System of Medicine* contained a modern account by G. Vivian Poore. Poore's account has been revised and modified by Head in the current edition of Allbutt's text-book, to which the reader may be referred.

General Definition.—Oppenheim's definition is the one most generally quoted, running as follows:

By an occupation neurosis is understood a disturbance of innervation of the muscles, occurring only in certain complicated movements acquired by practice, of such a nature as to permit the same muscles to respond to the will in all other actions; thus, in the typical occupation neurosis, writers' cramp; the muscles paralyzed for the purpose of writing are found to be entirely mobile for the other purposes.

Occupation neuroses are neuroses incident to special occupations, and might better be termed vocational neuroses. They have been termed craft-neuroses (Head); but this term, perfectly suitable for writers' cramp, would

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nct so well fit a neurosis of preachers, namely, mogiphonia. Occupation-neuroses are neuroses of technique involving muscular action, and are well-nigh as numerous as are technical vocations themselves. It might be inquired whether certain apathies or neurasthenic phenomena could be regarded as a generalized occupation neuroses ("run down from overwork"); but this would be a doubtful usage of the term, since in such generalized disease the nervous system cannot be used effectively for the organism's general needs. The point of an occupation neurosis, on the other hand, lies in the fact that certain muscles, utilizable for all sorts of other purposes, can no longer be used for some special technical purpose in one particular manner learned by practice. Occupation neuroses are diseases of acquired function and as a rule of somewhat highly specialized function. Seldom do patients exhibit at one time two kinds of occupation neurosis; but a victim of writers' cramp may fall victim to typewriters' cramp and retain both.

It is a question what "neurosis" means in the term occupation neurosis. Occupation neuroses are, or should be, essentially functional neuropathies, in the most general sense of the term neuropathy, *i.e.*, either peripheral or central in origin. Theory tends to consider occupation neuroses as neuropathies of central rather than peripheral origin (Benedikt). But practice tends to find peripheral origins for many (G. Vivian Poore). Of course, if research should prove a peripheral (or central) structural basis for the clinical phenomena, then the name occupation neurosis would be itself a misnomer.

It is decidedly of more than academic interest to consider this question in individual cases. Consider in succession certain

Theories of Origin

1. Suppose, with a few authors, we consider occupation neuroses to be in part at least based on *constitutional factors or diatheses;* for example:

Féré, 1887–1900; Bonnier, 1898; Lanel, 1909; Head, 1910; Frankl-Hochwart, 1913.

Modern work has of course shown in all sorts of mental and nervous conditions, both functional, quasi functional, and frankly organic, that diathetic constitutional factors are fundamental and indispensable factors although they may not be the essential ones. The above-mentioned authors, in practically all cases, emphasize other non-constitutional factors in addition to the diatheses. The writers have histories available in a number of instances to show strong traces of hereditary factors, a finding which is not uncommon in the literature. This constitutional background naturally has important bearing upon the prognosis as well as upon general therapeutic measures.

Not only may the occupation neuroses be in a sense diseases of diathesis, but certain well-recognized diatheses may bring about phenomena which can

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with difficulty be distinguished from occupation neuroses; *e.g.*, tremor of old age, arteriosclerosis, tabes dorsalis, and dementia precox, yielding graphic disorder attributed by the patient and his friends to a functional effect of occupation. Parkinson's disease may occasionally suggest occupation neurosis. There are scattering references to occupation neurosis in gouty or rheumatic conditions.

2. We may also consider occupation neuroses to be in some way dependent upon (as it were, permitted by), *a general neurasthenic condition* which might be focalized in a particular muscular difficulty. The following authors touch upon this: Berger, 1885; Féré, 1887–1900; Bernhardt, 1896; Binswanger, 1896; Cassirer, 1900; Mettler, 1904; Lépinay, 1909; Starr, 1909; Vogt, 1909–1910; Head, 1910; Bing, 1912; Strümpell, 1912; Frankl-Hochwart, 1913; Jacobson, 1913; Oppenheim, 1913; Jelliffe, 1914.

The true neurasthenic should, as a rule, exhibit phenomena of more general distribution, involving numerous functions revolving about his complaints. The typical victim of writers' cramp will exhibit a complete, and indeed over-eager, desire to write, although his paralysis forbids the process; whereas the graphic impotence of the neurasthenic is coupled with a diminution of will or absolute aboulia.

3. The last few years have brought out a crop of theories according to which occupation neuroses are in some sense *hysterical phenomena*, perhaps based upon inhibitions in a Freudian sense. According to this account, a particular function, such as writing or managing a telegraph key, becomes impossible because a derived movement of an opposite nature with cramp, tremor, or ataxia has preempted the muscles so that they cannot perform the desired act. T. A. Williams, 1912, has expressed a hypothesis of this nature, but according to him the psychogenesis in these cases is not especially along sex lines. Other representatives of a psychogenetic basis, at least in part, are Berger, 1885; Cohn, 1897; Léri, 1900; Destarac, 1901; Brissaud, Hallion and Meige, 1903; Mettler, 1904; Meige, 1908; Lépinay, 1909; Vogt, 1909–1910; Head, 1910; Lewandowski, 1912; Mohr, 1912; Frankl-Hochwart, 1913; Strümpell, 1912; Thomas, J. J., 1913.

By no means all of these authors would class themselves among the Freudians, but they all to some extent lay stress upon functional bases for these conditions, conceiving that they may represent a form of psychoneurosis, if not always clearly neurasthenic.

4. A number of French authors prefer to class the occupation neuroses among the *tics;* for example, Léri, 1900; Destarac, 1901; Rudler, 1903; Bonnus, 1905; Meige, 1908.

J. J. Thomas also speaks of certain training exercises in writers' cramp as along the lines of treatment in tic.

5. Emphasis upon *overexertion* in the particular professional act has been laid by Althaus, 1870; Vance, 1873; Berger, 1885; Benedikt, 1897; Church, 1898; Mettler, 1904; Meige, 1908; Vogt, 1909–1910; Head, 1910;

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Bing, 1912; Frankl-Hochwart, 1913; Jelliffe, 1912; Thomas, 1913; Curshman, 1915.

Most of these authors combine the fact, or hypothesis, of overexertion with a variety of other explanatory schemes for the production of the neurosis; thus, overexertion may conceivably be muscular or neural and if neural, it might be peripheral or central in origin. But many of the above-named authors consider that the local use of the muscles in question has set up a myositis, bursitis, tenosynovitis, or other local change of a chronic inflammatory nature which could interfere with fine movements and still permit coarser movements to be produced. Such changes, which are doubtless not seldom found in cases that receive the diagnosis occupation neurosis, might well produce pain when muscles are used, particularly for finer coördinate movements.

Under this heading should be mentioned also the toxins of fatigue which Weichardt has described; and the work of Lee upon the production of certain substances in prolonged activity of muscles, such as carbon dioxide, paralactic acid, and ammonopotassium-phosphate, is perhaps in point. It is not impossible that future research may work out hypotheses along the lines of the hormones. Of course it is not necessary that the substances produced by muscular action or overexertion shall exert their effects upon the muscles in question, but they may act through the circulation or by way of tissue clefts upon the nervous system, notably upon the unstable surfaces of separation or synapses between the conducting nerve elements (Lee).

6. There has been a recrudescence of *arterial spasm theories* which, although given up largely for the explanation (after the fashion of Theodor Meynert) of melancholia and mania, are thought possibly applicable to the occupation-neurosis group. As leaning in part to such a doctrine may be mentioned: Goldflam, 1901; Massaut, 1901; Goldstein, 1901; Determann, 1905; Erb, 1906; Raymond, 1906; Kronenberg, 1908; Lanel, 1909; Lépinay, 1909; Head, 1910.

Nothnagel had already reported, in 1867, a case which he interpreted in this way. According to this account, the spasmodic form of writers' cramp would in some degree resemble the phenomenon of intermittent claudication. The spasm in question is conceived to be a local one. No extended application of the idea of arterial spasm as occurring in the *central* nervous system as a basis for peripheral effects has been found in the literature.

7. *Myotonia.*—Curschmann, 1905, has laid emphasis upon the close resemblance of cases of writers' cramp to myotonia. He has reported the case of a young brush maker whose partial myotonia closely simulated a professional neurosis with muscular weakness, paræsthesia, and spasm.

8. Under the influence of Duchenne and Benedikt, the majority of systematic writers, and a great many of the monograph and periodical writers, have supported what may be called *the central nervous theory* of origin. It may almost be said that such a definition as that given above from Oppen-

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heim really involves such a central nervous origin, although it is far from agreed as to the exact nature of the central dysfunction which gives rise to the dyskinesia. Among the supporters in part or as a whole of this account may be mentioned Giegel, 1864; Solly, 1864; Althaus, 1870; Benedikt, 1874, 1897; Beard, 1879; Seeligmüller, 1882; Berger, 1885; Bernhardt, 1896; Cohn, 1897; Church, 1898; Cassirer, 1900; Savill, 1901; Haskovec, 1902, 1906; Brissaud, Hallian and Meige, 1903; Mettler, 1904; Edinger, 1906; Gowers, 1907; Lépinay, 1909; Vogt, 1909, 1910; Head, 1910; Frankl-Hochwart, 1913; Binswanger, 1896; Duchenne, 1860; Kouindjy, 1904–5; Strümpell, 1912; Thomas, 1913.

Duchenne, in 1860, in his work "L'électrisation localisée," said that he defined as *spasm with functional impotence of muscles* certain affections characterized either by continuous contractions or by clonic contractions. The contractions might be painful, or they might not be accompanied by pain. The clonic contractions might appear in the form of tremors. The muscular impotence might manifest itself neither by contractions nor by pain, but solely in the exercise of certain voluntary or instinctive movements localized in the muscles appropriate to (synergic with) the movement.

Duchenne strongly leaned to the central view. He quoted a patient who suffered from right-sided writers' cramp and learned to write with his left hand, only to become victim to left-sided cramp. Duchenne felt that such cases would argue an extension of the morbid state from one side of the spinal cord to the other.

Other authors, notably Benedikt, take into account not the spinal cord alone, but the higher levels and notably the voluntary levels of the nervous system. Benedikt, 1874, was far more concrete than Duchenne in his hypotheses. He insisted that the cells disordered in writers' cramp must be associative cells rather than the cells governing single movements, for if the latter were affected, then paralysis must follow in such wise that none of the muscles in question could act. Benedikt assumed a condition of either (*a*) heightened irritability or (*b*) lessened irritability in a group of cells superintending the coördination of certain movements. In the case of heightened cell irritability, the normal stimulus from other areas (that is, by the "will") produces a cramp of the muscles. In case there is a lessened cell irritability, there will be a paralysis of coördination and the pen will drop loosely from the hand.

9. The modern work has conspired to produce a group of *peripheral theories* of the origin of occupation neuroses, but it is a question whether, if proof could be adduced for such theories, the entity or group of entities called occupation neuroses would not largely fall to the ground. That this would be the case is shown by the great emphasis now being laid, even in systematic accounts (*e.g.*, Head following the lead of G. Vivian Poore), upon actual or conceived local changes in muscles, nerves, their investments, or adjacent structures. Time and again in the course of preparing this syste-

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matic account we have been impressed with the notion that no such group of diseases actually exists, but that every concrete case deserving the appellation will turn into something far more definite than such a definition as that of Oppenheim's above would entail. Vivian Poore gave a number of reasons against regarding central disturbance of coördination as the basis of these so-called neuroses. Poore stated that he had never seen a case without evidence of peripheral change. In most of his cases, he had no evidence of disease except at the periphery. The disease at the periphery consisted of muscular paresis, muscular spasm, localized tremor, fibrillary tremors of certain muscles, alteration of muscular irritability, localized pain, nerve tenderness. Poore writes: "One or more of these symptoms was always present. The writers' cramp of the text-books, in which failure of the writing power is the sole symptom, I have never seen." In most cases the symptoms grew more or less gradually, and accordingly did not seem to be related to a sudden *débâcle* of coördination. Accordingly Poore thinks that writers' cramp belongs in a catalogue of diseases near neuralgia.

An American neurologist, Beard, also thought, in 1879, that the disease was a peripheral and local disease of nerves and muscles. He thought it became only secondarily, and rarely, a central general disease. Another American neurologist, W. E. Paul, also follows the tendency of Poore and Beard. Paul states that the "principal result of an analysis of these 200 (Massachusetts General Hospital) cases is that muscles, joints, and regions near joints are preponderantly the loci of sensory symptoms rather than the course and distribution of definite peripheral nerves." He goes on to say that "This, then, is my conception, that the impacts, compressions, squeezings, tensions, and stresses of excessive physiological functioning, acting on muscle tissue, or on sensory and motor nerve structures which run in the soft tissues and terminate in muscle, tendon, joints, or fasciæ, bring about neurolytic or myolytic changes responsible for the occupation neuroses and so-called occupation neuritis and occupation pain."

We have gone over a number of cases in local Boston clinics, and Paul's results at the Massachusetts General Hospital have been confirmed. No case has been found which entirely satisfies the general definition of Oppenheim. In the first 2000 cases in the Harvard Medical School Out-patient Clinic, and its continuation in the Out-door Department of the Peter Bent Brigham Hospital, the diagnosis of occupation neurosis has not occurred.

We feel that modern finer methods in local neurological diagnosis may serve to destroy the functional nature of other doubtful cases. In a case which might well be regarded as one of occupation neurosis, lately seen, a case which might be termed one of stonecutters' palsy, we were able to show long after the patient had ceased to complain of subjective sensory disorder a definite anæsthesia to faradism (Martin sensory tests).

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Varieties of Occupation Neuroses

A book of inordinate length would be required to deal with all varieties. Indeed, Vogt remarks that there is an endless number of occupation neuroses, as many as there are possibilities in handicraft activities and in varieties of technique. New types of occupation neuroses are developed as new handicrafts and techniques develop. The following is a partial list of occupation neuroses aside from the typical neurosis of all—namely, writers' cramp. These have been obtained from the literature, others could be added almost *ad libitum*.

Auctioneer
Bicyclist
'Cellist
Cigar roller
Cornetist
Clarionetist
Compositor
Diamond cutter
Dancer
Engraver
Enameller
Flutist
Flower maker
Gold worker
Harpist
Hammerman
Knitter
Locksmith
Lithographer
Lathe turner
Laborer
Letter sorter
Milker
Miners' nystagmus
Mason
Money counter
Microscopist
Nail maker
Organist
Pianist
Painter
Preacher
Shoemaker
Smith
Sawyer
Scissors sharpener
Sewing machinist
Singer
Seamstress
Sailor
Shaver
Trap drummer
Tennis player
Tailor
Tinker
Telegrapher
Trumpeter
Turner
Treadler
Tawer
Watchmaker
Walker
Zitherist

It is worth while to describe writers' cramp more in detail than the other occupation neuroses because it is more frequent than any other and because it has been more frequently described. Writers' cramp is still the most frequent form of occupation neurosis, although the number of cases has appreciably diminished since the advent of typewriters, which has of course entailed (but apparently in smaller numbers) a new form of occupation neurosis. Perhaps the best description of writers' cramp may be obtained from Gowers' text-book, although the disease is fully described in various places.

Writers' cramp is commonly stated to occur in twice as many males as females. It occurs in all adult ages, and Gowers found two cases between 10 and 20, 52 between 20 and 30, 50 between 30 and 40, 32 between 40 and 50, 12 between 50 and 60, and 5 in patients over 60. This series of 153 cases included 33 by Poore, 64 by Berger, and 54 of Gowers' own cases. There seems to be no special racial tendency to writers' cramp.

An hereditary tendency to nervous affections of various sorts has been found by certain authors. We have ourselves come upon an interesting example of familial writers' cramp. This case, a woman of 44, was a copyist, with a younger brother who has already at this time (aged 37) developed diffi-

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culty in his writing. The father of these two was likewise a copyist, and died at the age of 75 with mental symptoms and brain disease. The father's brother and sister also died in the seventies of shock, but neither they nor their children showed any trace of writers' palsy, although some of the children have been engaged in clerical work.

Among the general underlying conditions which favor the development of writer's cramp have been mentioned neurasthenia, a variety of diatheses and general conditions (gout, rheumatism, Bright's disease, syphilis, arteriosclerosis), overexertion, trauma, alcoholism, overuse of tobacco, etc. Emphasis has often been laid upon emotion, especially the emotion of anxiety, as a contributory cause. The Freudians are not loath to offer a sexual basis for certain cases. Some authors speak of influences lowering the tone of the nervous system, hypo-oxidation and the like; or the use of poor pens; or the continual improper holding of the pen; or even the use of poor paper. Somewhat more to the point probably are suggestions to the effect that painful affections of the fingers, neuritis, and neuralgia lie at the basis of certain cases. Much has been written concerning the art of penmanship with relation to the use of the larger muscles, and an excursion from the elbow and shoulder rather than from the wrist alone. It is thought that the employment of these wider excursions diminishes fatigue, on the basis that the smaller the muscles the greater the fatigue.

The typical case of writers' cramp, accordingly, is likely to appear in the male, but the figures signify rather that under European conditions more men than women follow clerical work for a living. Heredity will be found as a rule to be so vague as not to aid in the diagnosis. It is probable that the various "underlying conditions" above mentioned give rise to, or permit, the occurrence of neuritis rather than the true neurosis which classical authors signify by the term "writers' cramp."

1. *True writers' cramp* in the narrow sense of the term (neurosis minus local, peripheral, structural, or functional changes): Poore found 32 cases in a personal series of 75 which he was inclined to put in the group of true writers' cramp, but he states that, "In every case of impaired writing power that I have seen, there has been evidence, more or less marked, of derangement of one or more of the muscles used for writing." Poore's method of determining peripheral alteration in these cases was by artificially stimulating the muscles used in writing by means of faradism. He maintains that he found an excess of irritability in one or more of the writing muscles in most cases. Benedikt, in 1874, remarked that there was an increased faradic irritability in the earlier phases of the disease, whereas later phases were attended by a decrease. Benedikt, in 1897, remarks that the actually paralyzed muscles exhibit a decreased electrical reaction, whereas the neighboring muscles exhibit an increased irritability. It may be remarked in passing that, if these remarks of Benedikt are correct, then the innervation streaming down from the central nervous system with a perfectly normal

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intensity, rate, and periodicity might on account of local neuropathic or myopathic conditions produce the effect of ataxic writing, or even agraphia, without our being forced to the hypothesis of central changes. Other authors are not so emphatic in their statements concerning altered local irritability, although most concede a slight degree of quantitative change to the ordinary electrical tests.

The sub-groups of so-called true writers' cramp are, according to Gowers and most systematic authors, the following:

(*a*) Spastic.
(*b*) Paralytic.
(*c*) Tremulous.
(*d*) Neuralgic.

(*a*) *Spastic Writers' Cramp.*—The onset in the spastic form is gradual, as a rule, beginning with slight errors in penmanship, such as irregular strokes and marks due to unintended movements; the writer finds his pen being held overtightly; the total process of writing becomes slower; the tight holding of the pen augments to a genuine spasm, and the spastic condition, at first confined to a few muscles, spreads to more numerous muscles. In those few cases in which the spastic form of writers' cramp begins abruptly, Gowers states that the attack is ushered in by local pain.

The fully established writers' cramp in spastic form shows almost always tonic spasm interrupted at rare intervals by a slight accentuation of the muscular contractions in the form of a starting or jerking movement. The tonic spastic cases are often accompanied by local tremors.

There are more rarely clonic types of the spastic form. The clonus may be so severe also as to throw the pen across the room.

The startling feature of such a tonic or clonic situation in the writing arm of the patient is that the patient can at the same time perform quite properly and readily practically all other coördinative movements as well as simple movements which are innervated through the same peripheral nerves. The altered potentiality of movement is so sharply defined that certain subjects becoming unable to use a pen can still write with a pencil. It is historically curious that with the modern increase of writing and the introduction of the steel pen, it was common to hear it said that there was no possibility of writers' cramp when using the old-fashioned goose-quill, and that the whole new-fangled difficulty was due to the invention of steel pens. Research disclosed, however, that scriveners' palsy was also a phenomenon of the goose-quill era. Such movements as those of the hand grasp are naturally retained, and the force of the grasp is as a rule unimpaired, although occasionally somewhat weakened. The instances of local muscular wasting should probably not be considered to be in the class of true writers' cramp, even as long-standing end results. The data concerning electrical irritability are, as stated above, somewhat equivocal, but that doubt is probably due to the inclusion of

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neuritic cases among the supposedly neurotic ones in a series collected by various workers.

Cases which are for convenience termed "spastic" cases are nevertheless accompanied by more or less marked sensory symptoms. Indeed, sensory symptoms of some sort are seldom absent (and the sensory symptoms may indeed occur alone in some instances; see below). The most frequent sensory symptoms include, besides a general sense of distressing fatigue, definite dull pains often referred to various structures in the hand, especially in the metacarpal region and the wrist. These pains subside quickly when the effort to write is relinquished. There may also be local tenderness as well as tingling and paræsthesia. It is probable that cases of various severe pains running apparently in the course of the nerves, with local tenderness, belong more truly in a neuritic group of definitely structural origin, but may be referred to here because of their association with local spasms. There are cases in which the patient when not writing is subject to local pains. Such cases may be interpreted along lines of argument as above stated as phenomena in cases of local neuritis. Cyanosis has been stated to occur with such cases (Brissaud, Hallian, and Meige). (See Anomalous Group below.) The outcome of these cases is dealt with below. The milder neuralgic and tremulous forms tend to go over into the spastic form, as well as does the paralytic form, about to be described. In certain cases the spastic form passes eventually into the paralytic form.

(*b*) *Paralytic Form.*—This form is perhaps the typical *impotence fonctionelle des écrivains*. In this form the subject, after essaying the use of the pen, gets a feeling of weakness and is unable to bear hard enough upon the paper to make properly legible marks. It would be exceedingly valuable if more extensive observations could be made with the Kraepelinian writing apparatus as studied by Gross; this apparatus permits registration of the pressure and rapidity of writing. Kraepelin states that in the depressive cases of manic depressive psychosis, there is a slowing in speed of writing and a diminution of its size accompanied by a reduction in the pressure. It is probably true that numerous cases of writers' cramp bear a more general resemblance to depressive cases of manic depressive psychosis than they do to such conditions as the maniacal phase or the so-called catatonic phases of dementia præcox. In the latter conditions, Kraepelin's associates found such writing as was performed to be performed as a rule without alteration or punctuated by characteristic sudden changes and interruptions.

Berger has noted cases in which the functional disorder appears to start in the shoulder girdle rather than in the forearm and hand. The surgeon would be tempted to see in such cases instances of local disease, such as subdeltoid bursitis; the *psychiatrist* familiar with mild cases of dementia præcox might be tempted to the hypothesis of a local muscular hypertension interpreted as a phenomenon of catatonia. Such a condition would resemble either true *flexibilitas cerea* or the pseudo-flexibility of some authors. The

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shoulder placed in a certain posture would retain this posture in such wise as to defeat the proper exertion of the rest of the extremity. The paralytic form does not occur abundantly in the literature of writers' cramp, but is mentioned as an end phase in certain cases.

(*c*) *Tremulous Form.*—This form has been indicated above. The spastic and other forms of the disease often begin with tremors or show tremors near the onset. The writing in the pure examples of the tremulous forms is said to be not unlike the writing characteristic of paralysis agitans. The characteristic handwriting in paralysis agitans shows, at least in later phases, a certain periodicity, so that in writing a number of sentences the manuscript becomes tremulous several times only to return to normality. In the case of multiple sclerosis, on the contrary, the tremors in writing become more and more marked as the "intention becomes more settled." The productions of the tremulous form have, however, in common with multiple sclerosis, the fact that with the progressive execution of the task the ataxia becomes coarser. The victim of multiple sclerosis, however, does not give up through fatigue or through aboulia as does the victim of writers' cramp. One of the synonyms for writers' cramp, "local chorea," is in short due to the appearance of the tremulous form or the tremors found in several other forms. There is no basis in the literature for any special relation between writers' cramp and true chorea. Work with the Kraepelinian writing registration apparatus would be of special service in cases thought to be of this form.

(*d*) *Neuralgic Form.*—Perhaps the neuralgic form stands next to the spastic form of true writers' cramp in frequency. In this form evidences suggesting neuritis are not greatly in evidence. The local neuralgia of independent nature is often found spreading from a single point in the hand and to accompany movements, but the execution of all other movements is made without pain. Of course, if the pain were found following the course of a particular nerve, the chances are that the diagnosis of neuritis would be preferred. Associated with the pains may be paræsthesias.

In general it may be observed, concerning the above-mentioned subforms of true writers' cramp, that the movements may be accentuated when the patient is under observation. The examining physician has to take this into account when evaluating the severity of a given case. It must become evident from references in the above paragraphs that the differential diagnosis is not always easy; further data in this direction will be given below. Systematic examination of these cases should show no alteration of mobility or of the touch, temperature, and pain senses. Some experience of the writers with the Martin sensory threshold tests seems to indicate that at least in some forms of occupation neurosis this very finely quantitative test for faradic sensibility (perhaps more finely quantitative than any other sensory test so far described) indicates that in writers' cramp also we may find it desirable to resort to this method. It may well be that by means of it

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we shall be able still further to diminish the number of cases of "true" writers' cramp. If future researches are to yield eventually a small but distinctive group of true neuroses, it is possible or probable that they will be found in persons also otherwise "neurotic." The discovery of cases with increased reflexes, with vasomotor symptoms, with tendency to morbid apprehensiveness, to moodiness and variability of temperament, would tend to bear out such an hypothesis. Cases on a so-called neurotic basis, "hereditary" cases, cases in which there is overwork and undernourishment, night work, alcoholism, and sexual excess, are mentioned, and perhaps belong in this vaguely definable group of neuroses.

In addition to such general predisposing causes, Bernhardt mentions a number of other causes which doubtless contribute more to the growth of writers' cramp about to be described than they do to true writers' cramp. The groups to be described are of a more structural origin; accordingly cases of writers' cramp are said to follow injury, to be associated with Dupuytren's contracture, to be a partial phenomenon of neuritis, to be due to bad habits in writing, such as using the little finger for a pivot.

2. *Paralytic Writers' Cramp.*—This form is intended to be distinct from the subform of paralytic *true* writers' cramp, since the present form is a form in which the paralysis of writing is due to the pressure of aneurysm on a section of the ulnar nerve, to local pressure, etc. It is conceivable that alterations in fine movements may be brought about by these pressures and yet leave enough of the major functions of the arm intact to convey the impression that we are dealing with true writers' cramp. The diagnosis must of course depend upon other investigations than those of the writing power. Vivian Poore found in his series of 75 cases six in this group. In full blown cases, there should be electrical changes that might well distinguish this group from true writers' cramp.

3. Allied with the paralytic group just described is a group termed by Poore "*degenerative*," occurring in nine cases in his series. In this group, the first sign of the degenerative neural change is the impairment of writing power; but in cases of this sort, the functional impairment gradually or rapidly passes off, and is supplemented by impairment in coarser functions.

4. The *spasmodic* group, comprising five cases in Poore's analysis of 75, may be of congenital origin; and in three cases was found to be due to hemiplegia and in large part related to post-hemiplegic phenomena.

5. *Neuritic or neuralgic group,* forming 19 cases in Poore's series. Poore states that it is highly probable that we have to deal in this group with a mild form of neuritis, or at least some form of vasomotor irritability involving one or more of the nerves of the affected arm. The symptoms in these cases involve a wider area than the writing muscles. Sometimes the symptoms appear without any endeavor at writing. Characteristic is the appearance of tenderness of the nerves. Many of the patients, according to Poore, complain of a queer feeling in the arm, especially at night, with a constant

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desire to shift its position and an inability to make the arm comfortable in bed. Poore found in five of his cases the assigned cause to be excessive work with the hand. In eight cases he found history of previous strain or injury, in five cases worry and fatigue; in six cases he found vasomotor disturbance.

6. *Anomalous cases* often diagnosticated as true writers' cramp. Writing disorder of the syphilitic group of disease of the nervous system may sometimes give rise to this diagnosis. Possibly the case of Brissaud, Hallian, and Meige of so-called acrocyanosis associated with writers' cramp may have been one of early dementia præcox. The patient in this case, 16 years of age, made fantastic figures, and had in addition to some psychical peculiarities a marked cyanosis of his hands.

The disease usually lasts as long as attempts to write are persevered in; often when the patient learns to write with the other hand, the same disease sets in on the opposite side—at least in some cases. It appears that improper holding of the pen has been stated to be a cause of proportionately greater increase in the disease. Gowers has stated that the disease tends to involve more and more muscles in association with any general weakness of the nervous system which may exist. It seems to be the rule that sensory symptoms show a greater tendency to spread than motor. It is clear that the patient is subject to anxiety as to his writing capacity, involved as it is in his earning power, which leads to a piling up of the agony in the sense of the American slang phrase to the effect that the subject's actual disappointment is reduced to his being "rattled." It appears that in certain cases rest has been known to terminate the symptoms.

The general prognosis is stated by the books to be often unfavorable, particularly if the disease has lasted for some time. Gowers states that those cases have a better prognosis in which pain rather than spasm or paralysis predominates.

I. GENERAL EXAMINATION OF A SUBJECT WITH WRITERS' CRAMP

It seems well at this point to make some statement concerning the general examination of a patient with writers' cramp because the general principles of such an examination are suitable for examining victims of other forms of occupation neurosis. Following the general tendency of Vivian Poore to regard the disease as perhaps more likely than not to be really of structural origin, Henry Head has offered valuable paragraphs upon the principles of such a general examination. Head states that every patient complaining of an occupation neurosis should undergo a complete examination as if for life insurance. In other words, the patient's heredity, past and present history, to say nothing of his temperament and character, must be finically investigated. A routine neurological examination must be per-

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formed with a special search for wasting processes as well as tremors and disabilities of coördination. The examination must be so thorough as to rule out arteriosclerosis, renal disease, and various diatheses.

Head makes a great point of examining the functions of the two arms and of all the rest of the body, particularly its upper part, in the nude. Asymmetry in postures assumed at command may easily betray unilateral muscular weakness. Defective action of the trapezius, the levator anguli scapulæ, rhomboids, and serratus magnus is thus made manifest if it exists. The general tests for motility are then applied.

There follows a general examination of sensibility with particular attention to the possible tenderness of nerve-trunks. Head emphasizes the necessity of testing the tenderness of the coördinate nerve-trunks of the two sides because, as he says, every nerve-trunk normally shows some tenderness. The following statement from Head is interesting enough to quote:

"This examination requires care, because normal nerve-trunks are somewhat tender, and it is only by comparison of one side with the other that we can arrive at any useful conclusion. The patient stands facing the examiner, with both hands resting, palms upward, on the examiner's shoulders. It is important that the patient's arms should be really at rest, and as free from all muscular efforts as possible. The physician then examines the nerve-trunks of both limbs simultaneously by means of gentle pressure on the ulnars behind the olecranon, on the musculo-spirals at the inner borders of the supinator longus, and on the median at the elbows, on the inner side of the biceps tendon. The simultaneous examination of the medians can only be made if the examiner crosses his hands. If the examination of the nerve-trunks on the two sides be made simultaneously, there is greater likelihood of the pressure being equal; and if the examiner find a distinct difference in the tenderness of the nerves, he is justified in recording it as a symptom of importance. The patient is, of course, in ignorance of the object of the examination, and when the nerve-tenderness is well marked his response to a gentle pressure of the thumb is startling alike to examiner and examinee. This comparison of the two sides can only be trustworthy when the nutrition of the two is equal; if one arm be wasted, the comparison is no longer possible. The search for tender nerves is important in all these cases; and not seldom one of the palmar twigs of the medium or ulnar is found to be tender, or tenderness is detected along the course of the suprascapular, circumflex, or anterior thoracic nerves."

Careful sensory tests should follow with special reference to their differences in coördinate parts of the two limbs. The orthopedic group of disorders must be looked for in the bones, joints, and ligaments.

Many authors have mentioned differences in temperament in these cases. We may again quote from Head in this matter:

"The patient, still stripped to the waist in a warm room, should now be made to carry out the particular act of which he complains. If it be to write, give him a sheet of paper and a good pen, see that he has a comfortable chair and table, and ask him to write his name and address, and the date. A great deal may often be learned by watching his attempts to perform the deranged action. Occasionally nothing amiss is to be detected, except perhaps that the writing may be produced rather slowly. The patient may tell you that the effort to write causes pain; but, so far as any change in the mode of production or in the product itself is concerned, there is nothing amiss. This, of course, is important both in diagnosis and prognosis. At the other end of the scale is the man who is scarcely able to

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make a mark upon paper; whose efforts are accompanied by most tumultuous movements of the shoulder, who is obliged to fix his arm securely before beginning to write; who grasps the pen with all his might, and perhaps drips with perspiration as he makes an abortive attempt to sign his name."

Electrical examinations have been mentioned above. Reaction of degeneration is never found in uncomplicated cases of occupation neurosis. Diminution in galvanic excitability is said to be confined to the small muscles of the hand and to affect the first dorsal of the interosseous group more frequently.

Differential Diagnosis.—The diagnosis of a disease due to occupation seems to be an obvious matter in instances of writers' cramp. The difficulty lodges, not so much in placing the condition in the group of occupation diseases as in choosing the condition fundamental in the particular case. This latter choice usually lies between the supposition of a true neurosis in the sense of a disease not primarily due to local structural changes, and one or other forms of neuritis, neuralgia, myositis, myalgia, or other local disease. As our description has abundantly shown, the very nature of the disease is in question, and the description is split along the familiar lines of controversy between structuralists and functionalists in neuropathology.

The fact seems to be that the majority of all cases of writers' cramp (which are said by some to form nine-tenths of all cases of occupation neurosis) are cases which fall outside the pale of the true neuroses. It is well to concede that there may be a small group of truly functional cases, and in any event to remember that in accordance with well-known principles, the range of functional symptoms in a given case may exceed the range of the immediately structural symptoms. Treatment may be governed accordingly. Taking for granted that the case presenting itself for treatment has in practically all cases made a diagnosis before resort to the physician, the physician must launch his differential diagnosis in the first place in the direction of excluding constitutional disease in which the writing defect may be merely the most striking phenomenon of weakness because perhaps it affects the earning power of the subject. Assuming that such constitutional diseases as have been mentioned above have been excluded by the most careful general medical and neurological examination, it would seem wise to include a careful psychopathological analysis from the diagnostic point of view. The books have probably not emphasized sufficiently the psychiatric possibilities in this direction and particularly mild depressive states which may or may not be distinguishable in a given case from the phenomena of psychoneurosis. If constitutional disease, the common neurological affections and psychiatric conditions of a pronounced character can be excluded, the process of diagnosis must begin to deal more particularly with orthopedic conditions, such as osteitis, periosteal changes, tenosynovitis, synovitis, arthritis and various forms of myositis and other myopathy. The discovery of various degrees of these orthopedic conditions does not, according to some authors,

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always mean that the entire condition is an orthopedic one. It may accordingly be well to hold in mind that osteopathy or myopathy is part and parcel of a more general process involving also the nerves, and that any or all of these changes may be associated with, preceded by, or followed by neurotic changes in the sense of purely functional conditions.

The analysis must next consider the neuropathies, general and local. If we should adopt the triple classification of James Mackenzie of structural, functional, and reflex symptoms, especially structural, functional, and reflex pain, we must, perhaps, exclude the so-called reflex pain from consideration inasmuch as it has not been shown that conditions in viscera can lead to such a condition as writers' cramp. However, if central nervous conditions suitable for the production of reflex pain could be brought about by local disease in the central nervous axis, then it is conceivable that there might be cases of painful writers' cramp of central origin. We have not encountered such cases in the literature. The symptoms would then in general reduce in the sense of Mackenzie to functional and structural ones; probably in most cases there is a combination of these. Despite the tendency of the writers to look for psychiatric causes in this affection, we have become more and more enamoured of what might be called the peripheral hypothesis, and to agree with the general modern tendency to discover in these cases neuritis in greater or less degree. An enumeration of conditions which have been or may readily be mistaken for true writers' cramp bears out this general tendency.

First among the conditions which should if possible be differentiated from the true occupation neurosis is what may be termed occupation neuritis, in which there will be found sensory changes (tenderness of nerve-trunks, motor changes), paralysis, tremor, etc., and electrical changes (in the extreme case, reaction of degeneration). Neurasthenic and hysterical patients are among those who may eventually exhibit writers' cramp, and it may accordingly be difficult to pull apart the effects of the underlying general neurosis and the particular phenomenon of the cramp, particularly in hysterical cases in which there are so often unilateral symptoms. Among diseases which have given rise to errors in diagnosis may be mentioned multiple sclerosis, tabes—notably brachial tabes, hemiplegia of a gradual development (the thrombotic type), paralysis agitans, general paresis. A number of psychiatric conditions need mention since the writing disorder may emerge from them as one of the most striking symptoms: brain tumor, brain abscess, senile dementia, alcoholic mental disease, post-infectious psychosis, chorea, the melancholic and catatonic phases of certain mental diseases, congenital agraphias, and so on. Dupuytren's contraction, acroparæsthesia, and even Raynaud's disease may need to be thought of. A fundamental study of a differential diagnosis of these disorders would, in fact, lead to including practically the entire range of nervous and mental disorders, together with a large group of orthopedic and general medical disorders.

The Range of Treatment.—The range of treatment in occupation neuroses

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is wide, corresponding to the hypotheses adopted by authors concerning cases. On the general hypothesis of fatigue, a number of bits of apparatus have been constructed which permit writing to be performed by the use of somewhat different muscles. It is probable that no one of these pieces of apparatus has been extremely effective. To meet local conditions, special gymnastic movements have been prescribed, active and passive movements, as well as passive movements executed by the non-affected hand. Work with Zander apparatus and massage has been tried, as well as galvanic and occasionally faradic electricity. Hydrotherapy has come in for a share of attention. Bier's hyperæmic method has been employed. The relief of irritation as by counter-irritation, and the use of the cautery, have been counselled, as well as muscle stretching. There is a large group of hygienic counsels; attention to the eyes, condition of the nerves and condition of the muscles, schemes for removal of the difficulty by suggestion or by subtler methods of psychotherapy and by psychoanalysis. Re-educational processes are mentioned, as well as the so-called education of the will. There is a group of recommendations beginning with rest. The use of the other hand, the use of the typewriter instead of pen and paper, and mirror writing (with the object of the correction of early acquired habits) are noted in the literature. There is a group of suggestions from the pharmacological side ranging from the local injection of carbolic acid (doubtless founded on the results in certain cases of trigeminal neuralgia with alcohol) to the exhibition of various tonics and other drugs. Both excitants and sedatives are mentioned; we have noted the following drugs: strychnine, atropine, bromides, narcotics, quinine, valerianate of zinc, arsenic, iodoform, gelsemin; zinc phosphate, Gowers is inclined to recommend above all the rest. He is willing to counsel writing with the other hand, although it is said in 50 per cent. of all cases the cramp occurs in the other hand after a time. Gowers thinks that stimulants are superior to sedatives in the general treatment of these cases, but naturally employs sedatives in various instances in which neuralgia is predominant.

II. OTHER MANUAL NEUROSES

First among these stands **typewriters' cramp,** a modern disease which comes on either independently, or after the therapeutic substitution of typewriting for manuscript which is occasionally counselled. Typewriters' cramp does not supervene immediately, but it appears that it may occur within a period of weeks or months after the handwriting has been given up. This disease appears to be extremely rare; Head has personally observed but one case. Dr. W. E. Paul of Boston has had a case. Typewriters' cramp is as a rule bimanual.

Telegraphers' Cramp.—This disease appears to be rare, whether in form of tremor, or paresis, or pain. One of Vivian Poore's cases may be abstracted as follows:

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A man, aged 39, had worked a Morse key for 19 years. He had as much difficulty in writing as in telegraphing, and it was noticeable that all delicate acts—such as shaving, holding a teacup, or wielding a salt-spoon—were seriously impeded by tremor. The tremor affected all the muscles of the right upper limb, including the pectoralis major; but it was most marked when the hand was prone in the telegraphist's position. The median and musculospiral nerves on the right side were distinctly tender.

This case might certainly be interpreted to signify a more general disorder than that in the hypothetic association sense for the manipulation of the Morse key. The literature contains a discussion as to whether the Morse key or the Hughes key is the more likely to bring about telegraphers' cramp.

Pianists' Cramp.—Cases of Poore, de Lépinay, and Riseau are mentioned. The literature indicates that the disease occurs in beginners rather than in virtuosi. A case of de Lépinay quoted by Head was that of a musician who at first could not extend the little finger of the right hand in playing the oboe. Later, the ring finger could not be extended, and trouble in playing the piano followed. Examination demonstrated that there was also difficulty in several other delicate acts performed by these muscles, to such an extent that it may be held that this case was one of musculospiral neuritis.

Violinists' Cramp.—The characteristic digital corns of the violinist occasionally become tender and cause pain that radiates up the arm and prevents firm pressure of the fingers on the strings. Such cases would have to be regarded as pseudo-cramp cases. True cases of occupation neurosis in violin players and players of other stringed instruments appear to be extremely rare.

Sewing Cramp and Scissors Cramp.—These are rare forms of manual occupation neuroses. The latter form appears under some circumstances to be due to actual neural and muscular atrophy produced by pressure from the scissors upon the thenar eminence.

Hammermen's Cramp.—Head points out that many cases of supposed Hammermen's cramp are really otherwise to be explained. Thus the hephæstic palsy of Frank Smith, Head regards as a cerebral hemiplegia or monoplegia occurring in a hammerman. Head believes, however, that there is a condition which approaches a true occupation neurosis, or is in any event a distinct clinical entity.

"I have seen many blacksmiths and men accustomed to the use of a hammer who complained that they could not hold it in one or other hand. But in every case the dissability depended on the presence of pain in the neighborhood of the elbow, evoked by attempting to grasp anything firmly in the hand. This condition seems to be a distinct clinical entity of frequent occurrence, but, so far as I have been able to discover, it has not been definitely described. Many cases of "hammermen's cramp" seems to have been instances of this disease, which I believe to be closely allied to meralgia paræsthetica in the leg (Bernhardt's disease).

"The patient begins to complain of pain situated over the insertion of the long extensors

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of the fingers just below the external condyle. The pain is never entirely absent, but is greatly increased when he attempts to grasp with the hand of the affected extremity. Should the patient's occupation necessitate any movement in which the long extensors participate, he will complain of professional disability. Thus, I have seen a blacksmith, a ladler of metal for stereotyping, and a Government clerk, all of whom suffered from this complaint; the first could not use his hammer, the second his ladle, and the third his pen, without bringing on the pain so severely that work became impossible. The Government clerk gave the following history of the onset of pain. Coming home one winter night from a week-end in the country, he arrived at the station to find no cab. He was therefore obliged to carry a heavy bag some distance to his home. Next morning he noticed pain over the extensors of the forearm, which was easier when the arm was flexed. It was greatly increased by writing or by handing a plate; grasping the spade while gardening produced severe pain. It gradually improved to such an extent that he was free from pain unless he wrote for long periods or performed any act which necessitated grasping. In these cases the pain seems also to produce a curious inhibiting effect upon the strength of the grasp, so that the patient complains that his hand 'feels weak;' and yet there is no weakness of any muscles of the hand. Extension is both painful and difficult, and so the wrist and fingers are no longer fixed, and the flexors cannot act for want of their normal support. This was well seen in the ladler of metal, who was no longer able to employ his right hand as a fulcrum when using his long handle.

"I believe this condition is produced by injury to the nerve-twigs which pass through the extensors to come to the surface just below the external condyle. This spot is intensely tender to deep pressure, and there is generally a patch of tenderness to the point of a dragged pin, with or without a little diminution in cutaneous tactile sensibility, over a patch a few inches in length situated just below the external condyle. No other nerve trunks are tender, and the muscular reactions do not shew any change.

"Complete rest of the arm with blisters applied to the tender spot will generally cure the disease in 6 weeks. But it is liable to drag on if the arm is used, and may fall into a chronic intractable condition."

Granite Cutters' Cramp.—We have had the opportunity of examining a case of cramp in which an apparent occupation neurosis was produced at the end of 15 years' of work with a pneumatic tool used in quarrying. This tool caused considerable jarring of the arm. The patient when seen had had 6 months of symptoms. The first symptom was pain in the forearm, radiating back to a point above the elbow. The pain had grown progressively worse, and the patient had thought of quitting work owing to a real or imagined difficulty in grasping the hammer. The patient had ceased work with this tool, 4 months before our observation, but the pain had continued. The case was complicated by worry over the payment of bills and the possibility of insurance. Wassermann reaction was positive. We found that a pain in the elbow would appear when the hand was flexed. A similar ache would appear at night. Early in the disease, the patient had had a certain degree of numbness of the ring and little fingers. The patient found that he could flex his forearm if in the full position of supination or pronation; but that, if the hand was held in a semi-prone posture, he could not flex his forearm without pain. This semi-prone position of the arm was the position characteristically maintained while working with the pneumatic tool. Examination of the sensibility of the skin gave a negative result to ordinary tests,

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nor was there any abnormal tenderness over nerve-trunks. The grasp of the left hand stood to that of the right as 42 to 38. We were, however, able to demonstrate by means of the Martin sensory threshold test for faradism, that there was still a slight degree of anæsthesia of the hand, most marked in the thumb and little fingers. It is, of course, possible that this electrical anæsthesia is in some sense functional, but it seemed more likely that it depends upon a structural neuritic disorder. Our experience with this case gives an intimation of what finer examinations may bring out in various cases of other forms of occupation neuroses, notably in writers' cramp. Cases in which no anæsthesia can be demonstrated with the methods ordinarily available to the clinician may yield results with this test. Upon inquiry, we learn that stonecutters not infrequently show a disease of this general character.

Cigarmakers' Cramp.—In Boston, and doubtless elsewhere where cigar-making is an habitual occupation, cases of cigarmakers' cramp appear from time to time. They have come to the attention of the Industrial Accident Board in Massachusetts, but it cannot be said that the condition has as yet been reduced to a well understood entity. We had the experience of a case characterized by a somewhat curious feature; just prior to each of several attacks of mania in the psychosis termed by Kraepelin manic depressive insanity, difficulty in rolling the tobacco leaves appeared, being the first sign or species of aura for these attacks.

Artists' Cramp.—A form of disease doubtless not truly belonging to the occupation-neurosis group was observed by us in a painter, who was the victim of a psychoneurosis characterized by irritability, hypochondria, insomnia, and an inability combined with a persistent desire to work; with the onset of the aboulia, the pain and incapacity to use the brush would occur. The artist would then give up his work for a time. Upon recovery from the psychoneurosis under treatment by means of rest, hydrotherapy, and argumentation, the patient entirely recovered his capacity to use the brush.

III. OTHER FORMS OF OCCUPATION NEUROSES

There is a small group of what might be termed pedal neuroses, of which may be mentioned dancer's cramp, the most frequent or only form of which known is that in the toe dancer. In the list given above are several examples of still other forms, such as buccal, laryngeal, and the like.

IV. GENERAL PROGNOSIS AND TREATMENT

It must be obvious from the above account that the diagnostic problems of the occupation neurosis group are almost inextricably mixed with those of various neuropathies and myopathies associated with different forms of occupation. Accordingly the most essential problem to solve is that of diagnosis

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in a given case. The experience of almost every neurologist is likely to be rather meager in this field. The careful investigation and report of all cases of occupation neuropathy and neuritis may be recommended, so that as the years pass a more and more comprehensive, systematic study of these allied conditions may be made. As in the case of stonecutters' disease mentioned above, it is possible that the Wassermann reaction may be of service in elucidating certain tendencies to neuritis. And it may be here that such a theory as the *Ersatz Theorie* of Edinger may have its application. According to this theory, functional overuse or a part may for intimate metabolic reasons produce or permit structural changes.

As also noted above, a number of cases disappear from the occupation neurosis group and reappear in the group of the frank psychoses.

Aside from various pseudo-forms of occupation neurosis (of which a sufficient list has been given under the heading, "Writers' Cramp"), the true occupation neuroses form a group whose prognosis is on the whole not good if we make it our aim to send the patient back with his original capacity to perform the function with which he now has difficulty. The prognosis as to a perfect recovery of the original function is decidedly not good. Also, in sundry instances, the prognosis is unfavorable with respect to the success of a substitution of some nearly allied function, such as typewriting for manuscript. On the whole, it would seem wiser to take steps at once to change the occupation somewhat radically if the diagnosis of true occupation neurosis can be made reasonably certain.

As to treatment, the general counsel of rest from the occupation in question is well-nigh universal. Naturally, if the diagnosis be incorrect, the counsel "rest" may be also decidedly incorrect.

Substitution of a new form allied to the old is the second general prescription. It would seem very unlikely that there should be an endless chain of disabilities ensuing upon substitutions of new forms of occupation unless there is some more fundamental form of disorder at the bottom of the condition, such as, for example, a psychoneurotic or frankly psychopathic condition. In a country like the United States, wherein it seems to be almost the universal habit to change occupations more or less frequently, the securing of substitute occupations is perhaps not so difficult as in a country of more stable social conditions. We hardly possess in the United States such conditions as hold in the greatly extended civil service of Great Britain.

If, however, rest, with or without a substitution of a new form of occupation, cannot be executed, or if the disability is such as to be, as it were, more irritative than destructive to the performance, then it might be inquired whether gymnastics and exercise would not be of benefit. It does not appear that in cases of true occupation neurosis gymnastic exercises have been of any benefit.

A list of drugs used in writers' cramp has been given above and need not be repeated here. The best general injunction accordingly in a case of sup-

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posed occupation neurosis is that the diagnostician shall endeavor by all means to prove that case is not one of occupation neurosis. The chances are very good, as the above account has intimated, that the expert diagnostician will prove that the majority of these cases are actually neuritides, or examples of neuropathies, myopathies, arthropathies, or other local forms of disorder affecting finer acquired habitual movements. Thus, the same general tendency which we observed in neuropathology at large obtains in this field also, namely, that the neuroses in the sense of functional conditions tend to disappear into conditions in some sense structural. To be sure, the structural conditions in questionmay not need to be overt examples of nerve or muscle wasting or of microscopically demonstrable fatty degeneration, but the technique of the future will doubtless prove able to demonstrate finer forms of physical and chemical disorder which will serve better to explain both neuroses in general and the small but perturbing group of occupation neuroses.

A note should be made of a new and practical phase into which the occupation neurosis question passes through the enactment of various workmen's compensation acts. Problems of a most intricate nature come up in connection with arbitration boards dealing with these questions. The whole topic of simulation enters to obscure the issue. It is too early to speak of the practical effect of these acts in this country. It is probable that the theory of the condition will be greatly benefited by the careful expert examinations which will be performed in these cases. We have already noted a tendency to greater care in these examinations as a result of the work of a commission like the Industrial Accident Board of Massachusetts.

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DIVISION IV

Occupational Affections of the Nose, Mouth, Throat, Eye, and Ear

CHAPTER I

OCCUPATIONAL AFFECTIONS OF THE NOSE, MOUTH AND THROAT

BY T. M. LEGGE, M. D., London, England

I. The Effect of Dust and Fumes on the Upper Air Passages

The bichromates are one example of corrosive chemical action causing acute inflammation of the upper air passages and progressing to a definite pathological lesion in perforation of the septum of the nose. Almost precisely similar corrosive action is exhibited after inhalation of the intensely irritating dust of white arsenic, or of compounds into which arsenic largely enters, such as arsenite of copper (Scheele's green), aceto-arsenite of copper (emerald or Paris green), and a mixture of white arsenic, caustic potash and flowers of sulphur largely used as a sheep dip. As to the action of these the reader is referred to the chapter on "Arsenic Poisoning."

Dusts of a hygroscopic nature also are known to cause inflammation of the nasal mucous membrane accompanied not infrequently by perforation of the septum. Examples of this are sodium chloride (common salt) and the chlorides of calcium and magnesium. Among 165 persons employed in salt grinding and packing, Müller found 45 suffering from catarrh of the mucous membrane and 42 with perforation of the septum. Cement dust similarly seems to exert nearly all its effect on the upper air passages (causing occasionally perforation of the septum), leaving the lung tissue practically unaffected.

Any general consideration of the action of dust on the upper air passages, however, must take account not only of the chemical composition but also of quantity, quality (whether organic or inorganic and, if the latter, whether metallic or non-metallic), and size and shape of the particles. The effect of gases and fumes which we are considering in this relation depends similarly on their chemical nature and on the concentration in which they are present in the atmosphere breathed.

Dealing first with dust it is safe to say that no dust, if inhaled in inordinate amount, can be without effect on the mucous membrane of the upper

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air passages. And by inhalation in inordinate amount is meant dust which can be seen constantly rising to the face of the operative in any manufacturing process. The effect of such inhalation is to produce a hyperæmia of the blood-vessels and frequently an eczematous condition round the alæ nasi, and to keep up a constant irritation of the mucous membrane lining the nasal passages, which leads first to hypertrophy with discharge of mucus (the signs of chronic nasal catarrh) and passes on eventually, in the course of years, to atrophy showing itself in pallor of the mucous membrane and loss of vibrissæ. The subject becomes a mouth breather. These evidences I have invariably noticed in workers wherever the air has been charged with fluff or dust, as in such occupations as fur pulling, cotton-waste sorting, flannelette raising, and all kinds of polishing operations on a wheel. The harder and more gritty the particles of dust produced are, as in metal grinding, sandstone cutting and the like, the more rapidly and surely will the condition described be produced. My colleague, Dr. Collis, after examining thousands of grinders and granite cutters and others exposed to inhalation of dust in Sheffield, Aberdeen and elsewhere, found, as a rule, that the lining membrane of the interior of the nose for a distance of ¾ in. was smooth, dry and pale colored; the mucous membrane behind this was red and inflamed and generally covered with dust, while the back of the pharynx and the pillars of the fauces were tolerant of the touch of the spatula used to depress the tongue, having lost their sensitiveness.

The action of ordinary dust, therefore, on the upper air passages is a mechanical one. Repeated irritation and pressure caused by deposit of dust on the septum may lead to ulceration passing on to a small perforation. Such perforation—very different from that set up by arsenic and bichromates—I have observed in metal grinding and cotton-waste sorting. Associated with this mechanical irritation arises not infrequently, by extension along the Eustachian tubes, inflammation of the middle ear resulting in partial deafness. The inflammatory process in the nose is continued down into the pharynx, eventually leading to atrophy of the mucous membrane as a result of the chronic pharyngitis set up.

The distinction between the effect of dust upon the upper air passages and upon the lungs is a narrow one. It would be out of place, however, to deal here with asthmatic, bronchitic and fibroid affections of the lungs, except to say that the researches of Dr. Collis bring out the fact that dusts of vegetable tissue, especially such fine dust as is given off in cleaning cotton-carding machines ("card stripping") and in the early processes of heckling and carding flax, hemp and jute, give rise to chest affections which may be described as asthmatic, while inhalation of mineral dusts containing free silica gives rise to fibroid phthisis in proportion as the free silica in the dust increases in amount. Animal dusts appear to have less injurious action than either vegetable or mineral dusts.

As to the mucous membrane of the mouth, mention only need be made of

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the blue line on the gums caused by absorption of lead sulphide, and of the intense inflammations set up by inhalation of mercury vapor or dust and of fumes of white phosphorus, as they are all dealt with at length in special chapters. Inhalation of lime dust in the unloading of such cargoes as calcined spathic ores, in addition to severe inflammation of the nose, gives rise to development of small ulcers in the mucous membrane lining the buccal cavity.

Mention also need only be made of the extremely irritating effect on the mucous membrane of the respiratory tract of gases such as chlorine, sulphur dioxide and ammonia, and of vapors such as are given off from hydrochloric, sulphuric and nitric acid. Chlorine gas in the air breathed to an extent of only 0.001 per cent. is highly injurious to man, causing cough, dyspnœa, bronchial catarrh and, if excessive quantities are breathed, great respiratory distress and rapid death. Hydrochloric acid fumes, on the other hand, in the proportion mentioned are much less irritant. When the proportion reaches 0.005, toleration becomes difficult. Nitrous fumes are a grave source of poisoning—if such a term can be correctly applied to a gas setting up, when inhaled in concentrated form, inflammation of the lungs with all its accompanying symptoms of respiratory distress. Onset of the acute symptoms is often delayed for from 12 to 24 hours after inhalation of the fumes, the workman meanwhile being able to continue at his work. The explanation of this is that time is needed for the inflammation to bring about the exudation into the smaller bronchial tubes and air vesicles which may eventually completely fill them.

Hydrofluoric acid causes, even in weak solutions (0.02 per cent.), irritant, symptoms of catarrh of the mucous membrane of the respiratory tract, lachrymation, etc. Strong solutions set up obstinate ulcers, difficult to heal, in the mucous membrane and skin.

In considering preventive measures against inhalation of dust, precedence must be given to its removal by locally applied exhaust ventilation as, unfortunately, the wearing of a respirator is neither in itself a sufficient protection nor, if it were, could the constant wearing of one be enforced. The conditions which a respirator should fulfil to be effective are, first, that the air breathed is freed from dust and, secondly, that it should not give discomfort to the worker. Further, it should be simple in construction, easily applied, and allow of frequent cleansing or renewal of the filtering material. As a matter of fact the more closely to the face a respirator is made to fit, and the more effectually it filters the air breathed, the greater is the inconvenience experienced when it is worn. This is shown in increase of the respiratory movements and pulse rate brought about by the exertion involved in aspirating the air through the filtering medium, and unavoidable rebreathing of some of the expired breath. Respirators, therefore, except for work lasting a short time—half an hour to an hour—cannot be considered a sufficient means of protecting the worker against dust.

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II. Chrome Compounds and Their Effects

Chromic acid and the bichromates of potassium and sodium are used in a number of important industries and may cause peculiar lesions, namely; erosion of the septum of the nose from inhalation of the dust, and eczematous eruptions or ulceration of the skin when abrasions allow either the crystals or solutions containing them to penetrate below the epidermal layers of the skin.

Manufacture.—Potassium bichromate is made on the large scale by roasting a mixture of chrome ironstone, potash and lime, lixiviating the fused mass with water, and adding enough sulphuric acid to convert the neutral chromate into bichromate. The crystals form beautiful adherent masses on the sides and floor of the crystallizing vats. They are broken up by a pick, removed on barrows to be washed, dried in open stoves by hot air or steam pipes, and finally packed in barrels. Sodium bichromate is made in practically the same way, sodium carbonate taking naturally the place of potassium carbonate. In the packing of the sodium compound there is little or no dust owing to the hygroscopic nature of the salt, whereas with the potassium compound elaborate exhaust ventilation is necessary to prevent its dissemination into the air.

Uses.—The bichromates are much more used in the arts than the chromates since they are the richer in chromic acid. Their principal uses are:

1. In the manufacture of the well-known chrome yellows, oranges and reds, by interaction of a solution of lead acetate and a bichromate; the chrome yellows by mixture with Prussian blue yield the greens called "Brunswick" greens. In their manufacture in addition to the risk from chrome there is the still greater one of lead poisoning.

2. In dyeing and calico printing. In dyeing cotton yarn an orange color the hanks are first soaked in limewater and transferred, after wringing, to a vessel containing lead acetate. They then pass through a dilute solution of a bichromate which develops the yellow color on the fiber. Treatment differs according to the particular shade of yellow color desired. Danger of lead poisoning has to be carefully guarded against from the dust given off in the "heading" or shaking of the dried hanks over a post, which can be secured only by exhaust ventilation carefully applied to the heading post.

In calico printing potassium bichromate is used in the discharge style for indigo blue and Turkey red, when it may be printed from a paste containing 40 per cent. of bichromate which will discharge the color from the blue or red material after suitable treatment. Or it may be used for the production of chrome lead colors by first printing the desired pattern on the calico with a paste containing acetate of lead, and subsequently passing this through a 2 to 5 per cent. solution of bichromate.

In aniline black dyeing similarly, after treatment with aniline hydrochloride, the calico cloth is passed through a bath containing dilute solution of bichromate.

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3. In photography and litho-etching. The carbon process depends on the fact that gelatine and potassium bichromate combine under the influence of light to form an insoluble compound. Thus the gelatine under the transparent portions of a negative when exposed to light becomes insoluble, retaining the pigment, while the darker parts are unacted upon and may be dissolved in warm water.

4. As an oxidizing agent for the manufacture of coal-tar colors, especially of anthracene to alizarin, and, in solution with sulphuric acid, as a bleaching agent for oil, tallow, etc.

5. In tanning leather by the "two-bath" process. This process which in the last 15 years has come into wide use requires special mention as it is a frequent cause of chrome ulceration. In the "one-bath" process basic chrome salts are used—chrome alum, etc.—which do not affect the skin. The "two-bath" process involves treatment of hides or skins in two distinct solutions generally known as "chromic acid bath" and "hypo" bath. The skins are first treated with a solution of chromic acid made by the action of hydrochloric acid on potassium bichromate (sometimes 4 per cent. bichromate and 4 per cent. acid on the weight of the pelt is used) and afterward with a solution of slightly acidified sodium thiosulphate to reduce the chromic acid to a basic chromic salt which produces the tannage. Risk of chrome ulceration is greatest in the operation of removing the skins from the first bath, spreading them out on a wooden horse to drain, and subsequently striking out the excess liquor—usually by machines carrying the skin upward between rolls, but sometimes by hand on sloping tables. H. G. Bennett in his book on "The Manufacture of Leather" says: "Equally good leather is produced by the "two-bath" and "one-bath" processes, but the latter is the cheaper process and is usually more convenient to manipulate. It has also the advantage that it does not give rise to the painful chrome sores which are very liable to occur with those who work in chromic acid liquors. The "two-bath" process is usually considered to give a more mellow tannage and a better color than the "one bath." . . . Broadly speaking, it is more suitable for the lighter leathers." The advantages of chrome tanning seem to lie in the quickness of the process so that the tanner has a larger turnover than with vegetable tanning. Hyposulphite of soda is recognized as useful in the treatment of chrome sores and use can be made of this fact by transference to work on the "hypo" bath of those suffering from chrome holes.

6. In French polishing, especially for darkening mahogany and walnut wood, the grain of which is brought out more clearly by bichromate than by any other substance. The liquid is applied generally with a cloth—more rarely with a brush.

7. As an ingredient of safety matches and in the preparation of colored glass and porcelain.

Effect on the Skin.—There is consensus of medical opinion that bichromate solutions do not attack the unbroken skin. The slightest break in

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continuity, however, especially where this is thin, as over the knuckles or between the fingers, suffices to start the destructive process which, once it has begun, will penetrate gradually through the soft tissues unless means are taken to prevent further contact with the solution. The seat of election for these sluggish ulcers or "chrome holes," as they are called by the workmen, is either on the knuckles or at the root of the nail, but they may occur on any part of the hands, forearm or foot. Where contact has been with dust I have seen them on the neck and in the groin. The tissues around the circular ulcer are heaped up, thickened, indurated and always undermined; the center is filled by a slough. When the slough has been removed the floor of the ulcer is seen to consist of grayish-yellow granulation tissue. In the majority of cases the diameter of the ulcer is not more than 1/8 in., in diameter, in a few it is 1/4 in. and the largest I have seen measured 3/4 by 1/2 in. The number of active chrome holes to be found on the hands of some workers when supervision is lax is surprising. In one man employed as a breaker in the crystal department of a bichromate works I found four on the right forearm, ten on the left, two in the groin and one on the back of the neck. In another man, similarly employed, chrome holes were found at the matrix of the nail of the index-finger of the right hand, and ring and little fingers of the left hand; the index-finger was much swollen and inflamed; on other parts of the hands also chrome holes were numerous. It is unusual not to be able to find small ulcers or the scars of ulcers that have healed on the hands of those employed in chrome tanning and in dyeing. Fortunately, to-day, penetration into joints and loss of fingers, described by early writers on the subject, are very rarely seen. Sometimes instead of taking the form described of localized ulcers, chromic acid in solution, as in the "two-bath" tanning process, sets up, in susceptible subjects, an extensive papular eruption on the hands and forearms, and occasionally on the feet and ankles also, accompanied by intense itching. In slub dyeing the chrome affection is found usually around the knuckles, on the palm, in the fold between the thumb and first finger, and about the wrist and forearms. The eczematous condition may extend to other parts of the body such as the face and back. Susceptibility plays an important part in disease of an eczematous nature the skin of some persons reacting much more readily to definite known irritants such as chromic acid than that of others. Experience can alone decide whether a particular person will be able to continue at the work or not.

Where the ulcerative process is serious it is impossible not to be struck either by peculiar susceptibility of the skin or by the evident want of care or ignorance on the part of the sufferers. New workers suffer much more than the seasoned who have experienced the painful condition. Lads of 14 and 15 years of age who start work in chrome tanning are sure to suffer if supervision is lax. The amount of pain and inconvenience caused by chrome holes is considerable. They are never a menace to life, but I have known six men absent in 1 year from one establishment for periods varying from 3

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to 9 weeks on account of them. Even when the severity is not such as to necessitate absence from work, months may elapse before they heal.

My experience dates back to the year 1899 when in consequence of the prevalence of chrome ulceration in the large bichromate works in Scotland employing 743 men, of whon 306 were engaged in processes involving exposure to chrome, I visited the factories and examined the workmen, taking note in each case of the age, duration of employment, precise occupation, condition of the septum after examination with the nasal speculum, sense of smell after testing with the essential oils of cloves and camphor, condition of the throat and eyes, and existence or absence of present or past chrome ulceration. In 39 out of the 176 men (22.1 per cent.) examined, one or more unhealed ulcers were found and in several others numerous scars marking the site of bygone ulcers.

In 126 men (71.5 per cent.) the septum of the nose was the site of perforation, and in 20 others of ulceration, in all probability a stage preceding perforation. Nearly all the men employed in the crystal department and at the furnaces showed perforation. It was noted as having taken place in one instance after a duration of employment of only 7 weeks, and in two others of less than 3 months. Usually it appears between the sixth and twelfth month after commencing work.

Nature of the Perforation.—The cartilaginous framework of the nose consists of five pieces—the two upper and the two lower lateral cartilages, and the cartilage of the septum. The two upper and the two lower lateral cartilages give the nose much of its shape and form the alæ nasi. The ulceration process due to bichromate dust never attacks them. The cartilage of the septum is somewhat triangular in form and thicker at its margins than at the center. The anterior margin, thickest above, is connected from above downward with the nasal bones, the front part of the two upper lateral cartilages, and the inner portion of the two lower cartilages. Its posterior margin is connected with the perpendicular lamella of the ethmoid; its inferior margin with the vomer and the palate processes of the superior maxillary bones. The seat of election for the perforation to commence is a point about ¼ in. from the lower and anterior margin of the septum; the ulceration extends in a direction upward and backward. This point is precisely that where inhaled dust alights and I have no doubt dust is the cause.

The limitation of the perforation to the cartilage of the septum is accounted for by the fact that the mucous membrane covering it is adherent, forming the perichondrium, and is far less vascular than the mucous membrane lining the rest of the nasal fossæ. Once the mucous membrane is destroyed, the blood supply to the cartilage is cut off, and necrosis ensues. The ulceration having progressed upward as far as the junction of the septum with the ethmoid and backward to the vomer, becomes arrested. Healing then takes place, the bone never being attacked, and the cicatrix usually

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becomes covered with an echthymatous crust of mucus. In no instance was the anterior of lower border of the septum destroyed. Consequently the rigidity of the parts is maintained, and deformity, so prominent in other ulcerative processes attacking the nose, is absent.

The onset of the morbid process is ushered in by sneezing and the ordinary symptoms of nasal catarrh. The pain accompanying the ulceration appears to be insignificant. At any rate it had never been severe enough to necessitate absence from work or to call for medical treatment. The only apparent inconvenience which results is the formation of plugs of mucus in the nasal passages.

Mucous deposits and white patches were occasionally noted on the pharynx, but definite ulceration was in no case detected. Asthma, noted by the French writers on the subject, Delpech and Hillairet, was found in one instance—that of the partner in one of the works. In his case there was a family predisposition to it, but the first definite attack dated from contact with bichromate dust.

Preventive and Remedial Measures.—In factories for the manufacture of bichromates Regulations are necessary, prescribing as their main provision periodical medical examination of the workers and treatment by the appointed surgeon of lesions contracted in the work. In addition, it is essential that some responsible person with knowledge of first aid should be appointed to treat daily such cuts and abrasions as occur. One of the surgeons carrying out this work writes to me as follows:

As chrome holes are usually produced from trifling or unseen abrasions about the hands I insist upon the foreman examining the hands of the men daily in order to see if there is any irritation of the skin and, if found, to have the parts thoroughly washed and afterward protected by dressings. This recommendation, I consider, is important in that it impresses on the men the benefit of checking at once tendency to the development of chrome holes. They can be developed in a day although the escharotic effect of the chrome may not be apparent for a day or two afterwards.

In order to seal up a chrome hole and prevent further contact with the liquor I use an ointment of zinc or borax on a small piece of lint to cover the hole. I cover the lint with gutta-percha tissue the edges of which I soften with heat from a taper or chloroform and then squeeze close to the skin. Adhesive plaster may also be used as a further protection over the gutta-percha tissue. Attention must be paid to see that the men are following out the treatment and that the sores are improving—otherwise there is no alternative but to remove the man from the chrome department until the wounds are healed.

Another surgeon after insistence on the importance of covering up the smallest scratches thus describes his treatment of chrome sores:

I apply poultices by night to clean off the gray slough, and to diminish the inflammation of the thickened edges. In addition, I endeavor by scraping to remove the gray slough from the floor and scrape also underneath the undermined edges. I pack the little hole with boracic lint or preferably with cyanide gauze, and so further stimulate and encourage healing. Healing, however, is a slow process and it is a very small hole which heals in less than 3 months.

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Instead of the poulticing other surgeons use boracic fomentations and apply yellow ointment of mercury, or clean with hydrogen peroxide solution and treat with an ointment containing ichthyol applied under an absolutely waterproof plaster. Washing with a 5 per cent. solution of sodium bisulphite has been recommended on the ground that by its means the chromic acid radicle is split up and rendered inert.

Where the hands come into contact with bichromate solutions, as in chrome tanning by the "two-bath" process and in dyeing, the first essential again is daily inspection of the hands and arms of all the workers and immediate covering over of the smallest abrasion so as to protect it against further contact with the solution. For this purpose collodion ("new skin") is invaluable, but if, as may be the case over the knuckles it is inapplicable a dressing under an impermeable waterproof plaster or closely fitting India-rubber fingerstall must be used instead. In chrome tanning India-rubber gloves reaching well up toward the elbow are a great protection, but the wearing of them does not make the treatment of abrasions unnecessary. I have found them generally provided and worn, but the retention of perspiration caused by their use may tend to soften the skin in some cases. Only where discipline and supervision are thorough would it be safe to throw aside the protection of India-rubber gloves. Substitutes for them such as leather gloves are in my experience wholly unsatisfactory. Periodic medical examination of persons employed in chrome tanning is useful as it ensures choice of proper dressings, is a check on the way in which the foreman exercises supervision, and will prevent chrome holes from causing prolonged absentation from work.

Lastly, smearing of the arms and hands with fat or with special ointments undoubtedly serves as a protection to the skin. The following is the preparation recommended by Levi, Chemist of the Pfister and Vogel Leather Company, Milwaukee:

Petrolatum (Paraffinum molle of the B. P.)	3 parts
Lanoline	1 part

Melt on the water bath or stove; when melted and thoroughly mixed, add 10 to 15 drops of 90 per cent. pure carbolic acid to every 400 grams, or 5 drops to every 4 oz. of the mixture. Pour into a glass or earthenware jar and allow the mass to solidify, when it is ready for use.

The application is as follows: Let the workman clean his hands and arms thoroughly with soap and water. Rinse with warm water and while still moist apply the ointment. Rub in well, so as to cover all exposed skin, for about 2 to 3 minutes. Then take a clean cloth and wipe dry. The skin will be left entirely dry and with no greasy feeling. Lanoline is absorbed by the skin and the petrolatum forms a light coating on the surface. The application of the two inert substances prevents the action of the chrome upon the

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surface and at the same time the absorbed grease prevents the action of the chrome, should the outer coating of petrolatum wear off.

My colleague Dr. Collis, struck by the remark of a chrome tanner that a dilute solution of mercuric chloride or solution of cyllin when used as a hand bath prevented the development of ulcers, possibly because the ulcer formation was due to microbic action, supervening on the action of the bichromate, has had trial given with fair success to an ointment composed of mineral lard 3 lb., paraffine wax 6 oz., and cyllin 3 oz. Mineral lard is used instead of lanoline to prevent oxidation by the chromic acid.

In a case of severe papular eczema arising from contact with ammonium bichromate used in photo-engraving application of equal parts of calamine and boracic acid lotions quickly proved efficacious. These are the cases, however, which are liable to relapse on fresh exposure and raise the question as to further continuance in the process.

The effect of chrome dust on the nasal septum, despite elaborate exhaust ventilation applied in packing the crystals, cannot wholly be avoided. The inconvenience caused by its occurrence is not sufficiently great apparently to induce the workmen to adopt precautions which might, if regularly used, be effective, such as, cotton wool plugs or smearing the septum with paraffine. The surest preventive measure is effort directed to reduce to a minimum generation of dust. Fischer states that daily use of an ointment composed of 50 grams zinc oxide, 22 grams olive oil, 18 grams liquefied ichthyol, and 10 grams balsam of Peru has caused the disappearance in from 3 to 4 weeks of signs of ulceration on the septum in those commencing work.

The Regulations in force in bichromate works in Great Britain emphasize the need for fencing of uncovered vats, and provision of meal room, cloak room, washing and bath accommodation, in addition to exhaust ventilation and periodic medical examination.

I agree with the conclusion of Fischer and Lehmann, who have recently carefully examined the conditions in the bichromate works in Germany (the latter having also carried out animal experiments as to the effects of chrome compounds), that no constitutional disease, and especially no disease of the kidneys, is induced by work in bichromate factories or in chrome tanning.

Manufacture of chromates of lead is attended with considerable risk of lead poisoning. Lighter shades are made by a cold precipitation process and the deeper shades of orange and red by boiling the ingredients—lead acetate, pulp white lead, bichromate of potash and soda and sulphate of soda—while barytes is added as the color is being made. Danger in the first method hardly arises until drying and grinding under edge runners, sieving and packing, are effected. The dust, when inhaled, is quickly absorbed, and carefully arranged, exhaust ventilation is necessary in all operations in which dust is generated. Vessels in which boiling is effected require to be hooded over and the hood connected up with an efficient exhaust draught. Grinding of chrome greens is a very dusty process and, although only 10 per cent. of

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lead may be present, poisoning will occur in the absence of encasing and exhaust ventilation. In an analysis of 225 cases of lead poisoning distributed according to precise occupation I found at least 10 per cent. were traceable to chrome colors.

Cotton yarn is dyed on a considerable scale with chromate of lead. Much of the yarn so dyed goes to Oriental markets and it is the orange chrome—that most heavily weighted with lead—which is most in demand there. Not a few outbreaks of severe lead poisoning have occurred among the workers—generally women—engaged in shaking out the yarn over heading posts—due, in every case, to a breakdown in the exhaust ventilation locally applied.

In both the manufacture of paints and colors and heading of yarn dyed with lead compounds, Regulations are in force in Great Britain prescribing periodic medical examination.

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CHAPTER II

A. OCCUPATIONAL INJURIES AND DISEASES OF THE EYE

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Certain industrial occupations involve particular risk to the eyes from acquired disease and intoxication or by reason of accidents. It may be said that each trade has its own eye diseases and particular forms of trauma, and in some the relation of the ocular damages to the risk encountered is very great indeed.

The employer has now to take into consideration not only the medical care and hospital service for injured employees and compensation of damages therefor, but in many states and countries is compelled by law as well as sentiment to put in safety appliances which have come to be known as money-saving propositions, as well as reducing the number of workmen laid up by reason of accident and disease.

The intelligent workman does not desire plenary damages, for health as well as sight is priceless, but he does now insist upon his work being made as safe and free from accident and disease as possible.

A very large proportion of the injuries occurring in trades happen to the eyes, probably as many in number as all other injuries, and as a workman needs good eyesight he is usually totally incapacitated by such a lesion.

The subjects to be considered may be arranged as follows, in which discussion I will deal mostly with the ætiology and prevention of such incapacitations:

I. Injuries from accidents.
II. Diseases due to occupations, involving excessive eye strain.
III. Diseases due to excessive exposure to light and heat.
IV. Toxicology or systemic poisoning causing loss of sight.
V. The estimation of economic damage.

I. ACCIDENTAL INJURIES

By far the larger proportion of these happen in the iron and steel trades where the workman is exposed to cuts, burns, bruises and flying foreign bodies. Indeed in shops, such as wire, chain, tool and machinery, as many accidents to the eye may occur in a year as there are workmen employed. Most of them are trivial injuries such as motes in the eye, laying the man up but for an hour or a day or two; others however, expose him to partial or complete loss of

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eyesight with total loss of earning power immediately following the accident or occurring months or even years afterward, as from sympathetic disease.

The cosmetic relations of these eye injuries are important, for a one-eyed man finds it difficult to obtain a job. A white scar on the cornea, or a squint, even though little or not at all affecting the vision may be the deciding power in rejecting a man who otherwise is well qualified.

ETIOLOGY OF INDUSTRIAL INJURIES

In the so-called dangerous trades, particularly those dealing with the *iron and steel industries*, workers in machinery, butchering work, and building trades, occur the larger number of ocular injuries. Among these are the miners of iron, copper and coal, the smelters and machine builders. Those that are most subject to flying foreign bodies causing wounds of the eye are foundrymen, machinists, turners, borers, boiler makers, fettlers, smiths and polishers; workers by the fire and heat, puddlers, casters, glass workers, etc., are burned by fire, ashes or iron and slag. Contusions of the eye from falls, blows and thrusts come to all classes.

In *agricultural pursuits* there are many kinds of injuries, especially from foreign bodies. In lands where the ground is tilled largely by hoes, as in the stony country of Switzerland, the breaking of these instruments against the rocks causes many such injuries.

In agricultural pursuits the causes are injuries by straws, by branches in the woods, in working about cattle and by farming implements. There were injuries from foreign bodies in the cornea and conjunctiva, perforating wounds, contusions from sharp or pointed objects, injuries from lime or dung, burns and insect stings. Cauterizations from the arsenate of lead have recently been reported in spraying orchards.

In the *building trades* there are injuries from iron, stone particles, and splinters of wood, and from instruments used in work; in *quarries and mines*, explosions of powder and dynamite. Injuries from lime are common among painters. In *laboratory workers*, foreign bodies, burns and scalds, and splinters of glass; in *glass blowing*, burns and formation of cataract; in *cabinet workers* injuries from the materials they work with, steel, wood and bone; in *turners*, injuries from wood, bone, ivory, and stone, blows from sharp knives, etc.

Generally the injury is inflicted by fragments flying off while the mechanic is striking a chisel or piece of metal, as a hatchet or hammer.

Solids or gaseous substances may cause conjunctivitis in people exposed to the inclemencies of weather, stone impregnation of the cornea in stonecutters, ribbon-shaped keratitis in hat makers, masons, steel grinders, opacities of cornea due to nitronaphthalin and aniline, keratitis of oyster openers and caisson laborers, cataract of glass blowers. We may probably give the most prominent place to grinders or to workmen in factories who are either

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constantly employed in grinding edged tools, or, as is common in this district, care for their own tools, and find occasion to go to the grindstone or emery wheel a number of times daily. In such trades foreign bodies are very prone to become lodged in the cornea. In the course of a day a grinder may get several foreign bodies in his cornea or many days may elapse without so doing. If the cornea of an old grinder be examined carefully with a magnifying glass, it will be frequently found studded over with nebulæ caused by damage from getting motes in the eye which, by frequent repetition, will dull the cornea and after a while will diminish the acuteness of vision. I have, however, seen many workmen with such scars who possess normal visual acuity. These nebulæ have been caused by small fragments of emery, or, more rarely, stone or pieces of iron. In localities where emery is used for glazing cutlery, such accidents are very common. To those, who have watched a grinder at work it is remarkable how any one can remain at the wheel without getting foreign bodies into the eye every few minutes. The shower of sparks which proceeds from the dry grinding wheel shows the large number of foreign bodies that come away. Dry grinding is certainly more dangerous as regards the eyes than wet grinding, as in the former the sparks fly freely and it is a chance whether they hit the man's eye or scatter about the room. The pneumatic fans which are required in many of the United States and in England, in consequence of the deleterious effect of the dust upon the health of the operators, protect the workman's eyes by drawing into themselves the sparks and particles flying from the wheel. In wet grinding the particles do not fly about so much; still the workman's face becomes bespattered.

In the great majority of cases the damage occasioned by mishaps of this character is not serious unless ulceration follows. Other accidents, of course, may follow upon the injury to the eye on account of the disability and lessened protection that is thereby produced. In the case of workmen who frequently get injuries to their eyes, and always where a workman or patient has lost an eye, I advise the securing of an accident policy on account of the greater danger of injury from the lessened functional ability.

By far the most serious eye accidents happen to men engaged in working in iron or steel. Particularly in iron manufacturing districts the majority of serious eye accidents occur from chipping or fettling iron and steel, in the lighter iron and steel industries as well as in the heavy trades, where armor plates are made and heavy castings of scores of tons. A very large proportion of the accidents are occasioned by what is called "chipping" and "fettling." "Dressing" is the name given in some parts of this process. This work consists in chipping the rough edges from steel and iron castings, ingots, and all kinds of iron and steel work, and, among other things, even large armor plates.

Castings of either iron, steel, or brass are the most dangerous to work upon, because the chippings fly about on account of the metal being brittle.

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It is very dangerous to chip castings in corners or where the chipping strikes the metal and rebounds. Chippings from the castings are about 1/4 to 3/4 in. long and very sharp. When thin plates are chipped on the edges, the chippings are sometimes 1, 2, and 3 in. long before they break off. All castings are fettled at the foundry, that is, the runners are cut off, and the plates where the metal has run at the joint of the moulding boxes are trimmed off.

FIG. 13.—Men at boring mill should provide themselves with goggles as at any time a chip is liable to injure the eyes. U. S. Steel Corp'n. (*Würdemann.*)

Whatever be the special kind of metal or steel to be fettled, the manner in which it is done is practically the same. A hammer and chisel or sate are used, and with these the roughness is removed. Frequently, also, while one man places the chisel, another, or even two others, will use the hammer and are called strikers. I understand that at works where, say, 1000 men are employed, 200 or more will be occupied more or less in chipping. Many men are frequently working close to each other, so that the danger is not only to the worker himself, but to those around. Passers-by are by no means infrequently the victims, and many blinded in this way have come under my notice. The chipper himself is often hit by the rebound of the splinter after it has struck perhaps the narrow angle of steel or iron upon which he may have

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been working, or some other object. It must be recollected, also, that in the process spoken of the danger is not merely from the iron or steel which is being operated upon, but there are three other places from which splinters may be, and actually are, given off and cause injury, namely: the hammer-head, the chisel-head, and the chisel-point.

The sizes vary from the most minute to others measuring some inches in length, and they may be thick or thin. The injury inflicted differs, of course, in accordance with the size of the missile and the force with which it is projected. The small fragments may be thrown off with such velocity that they penetrate the eyeball and become imbedded in its interior, in some instances passing through the eyelid before reaching the globe. The destruction to sight in this way is very large.

In the case of breweries and bottling works, particularly of bottling of aerated water, there is a comparative frequency of accidents from the breaking of the containers. A firm in Sheffield having several different factories employing from 2500 to 4200 hands states that in spite of the most careful enforcement of the use of masks, gauntlets, etc., in 1 year there were nearly 400 accidents. The number of bottles that burst in 1 year is very considerable; new bottles or syphons are twice as liable to break as the old ones. About 1 per cent. of the new and old bottles break when filled. Syphons burst less frequently but the explosion and danger is greater; about one in 5000 breaks in winter, while the percentage is greater in hot weather. The greater number of bottles break in the filling machine but there is practically no work in any part of the factory which may be regarded as free from danger.

Certain forms of injury are more apt to happen to some parts of the eye than others. Cuts and gashes occur mostly upon the anterior portion of the eye and its protecting organs; the lids, globe, cornea, ciliary region. Perforating wounds may go from the cornea and sclera through the bulb into the orbit and the optic nerve. Lacerations, tears, and the wounds from the bites of animals, are upon the more exposed parts, while shot wounds affect all portions of the eye. Flying foreign bodies or motes are usually found under the lids, though they may be impacted in the outer part of the bulb, especially the cornea. These consist of cinders, iron and copper splinters, the latter of which are often imbedded in the anterior, or even go to the posterior portion of the eye, remaining in the iris, vitreous, or the coats. The cornea most often contains foreign bodies, less often the conjunctiva and sclera. The effects of blows is most apparent upon the outer portions, the lids, brow, and then the bulb. In the eye itself the blow may cause tearing of the retina and chorioid, indenting the eye, and may push the lens away, causing detachment. Burns and cauterizations, as a rule, affect only the outer structures, the lids, cornea, and conjunctiva. The same may be said of burns from flame, the effect of lightning, electricity and sunlight, which may affect the lens and also the retina.

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OBJECTS CAUSING OCULAR INJURIES

On account of the great sensitiveness of the cornea, the formation of scar tissue and the danger of infection, a very small solution of continuity of this membrane is of great importance. All substances which come into contact with the surface of the cornea are apt to cause erosions. These may occur from the finger nail, pieces of straw and twigs. Cuts of the eye obtain from sharp instruments, such as knives, shears, glass; thin points of metal such as

FIG. 14.—Men pouring babbitt should wear goggles or a wire mesh screen covering the face, as at any moment an explosion is liable to occur. U. S. Steel Corp'n. (*Würdemann.*)

tin, and all cutting substances. Piercing wounds also occur from sharp points of metal, wood, and glass, which are round and thin, such as needles, pins, awls, nails, pieces of wire, forks and other table instruments, thorns, styles, or animal's claws; or those pointed, but not specially sharp instruments, as daggers, poinards, swords, sabers, bayonets, knives, steel pens; or those long triangular or irregular, as the points of compasses, sharp-pointed pieces of wood, pencils, sticks, parasols, walking canes, and umbrellas. Gashes are formed by knives, chisels, sabers, axes, daggers, etc.

Contused wounds are caused by either sharp or blunt objects of iron,

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wood or other stuff; by flying pieces of such substances as iron, nails, pieces of horseshoes, and all substances used in iron works; by files, pieces of wood, sticks, stone, shell, etc. Such may also occur by the horns and hoofs of cattle, the beaks of birds, which cause combined tearing and contusion. A large number of cases of whip-lash injuries have been reported. Lacerating wounds obtain through the above objects but are not particularly due to injuries by machinery, hooks, nails, or to the teeth and claws of dogs and cats. Bite-wounds, however, are generally due to dogs, except in rare cases of hunting accidents where they occur from boars, bears, and wildcats.

Foreign bodies either remain superficially or go deeply into the eye or orbit. They are carried by either the air or the wind, falling upon the eye, as cinders, particles of stone, coal or wood ashes, pieces of grain or sawdust—the so-called domestic accidents.

Industrial injuries are due to machinery or hand-work, and as a rule are from pieces broken loose from objects and go more deeply into the structure of the eye or into its interior. These are particles of iron, coal, copper, slate, stone, wood, bone, and those foreign bodies which are driven by explosions, such as glass splinters from exploding retorts, pieces of copper from caps or cartridges, etc.

Contusions occur from blunt or round objects, as a rule of large size; from falls on furniture, blows of fists, instrument handles, etc., or from objects which have been thrown with force, as stones, baseballs, tennis balls, snowballs, potatoes, apples, etc.; also from corks, glasses, concentric tops of bottles from explosion of the same.

Burns are caused by the flame itself, by the sun or electric light, from glowing rods and glass, by hot but not melted metal, by hot ashes, grains of powder, burns of the hair, from burning gas and hot steam, from hot water, oil, petroleum, pitch, etc. Cauterization may be due to acids, such as sulphuric, nitric, hydrochloric, phenol, vitriol, to alkalies and caustics, as soda and potash, to lime burns, arsenate of lead, and to methyl violet pencils and other substances.

BACTERIOLOGY

The aspergillus fumigatus is the only mould that has been found to cause infection of ocular wounds. The micrococci occur in the following order: the diplo-pneumococcus, streptococcus pyogenes, staphylococcus pyogenes, aureus and albus. The ozena bacillus, gonococcus, bacillus pyocyaneus, bacillus pyogenes, staphylococcus pyogenes, citreus and fetidus, micrococcus cereus, tetragenus prodigiosus, and bacillus of tetanus are less often found. To these perhaps may be later added the presently unknown microörganism producing sympathetic ophthalmitis.

Staphylococci are constantly found in the skin at the edge of the lid and sometimes in the normal conjunctival sac; streptoccoci and pneumococci are never found in the normal eye.

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DISPOSITION OF DISEASED AND ABNORMAL EYES TO INJURY

Weak sight disposes to bodily injury, because the individual does not see well enough to escape accidents, as well as specific injuries to the eyes.

Diseased and abnormal eyes, as in those suffering from senile degeneration and weakened walls of the blood-vessels, are predisposed to bleeding into the tissues from contusions and concussions, and to this condition persons suffering from arteriosclerosis, glaucoma, diabetes, leukemia, and renal diseases are likewise subject.

TRAUMATISM AS AN EXCITING CAUSE OF CONSTITUTIONAL EYE DISEASE

Traumatism may be the exciting cause of syphilitic iritis and it is possible that a corneal wound may be the point of entrance for an exogenous infection, or a syphilitic iritis may be superimposed on an iritis from other cause.

MECHANISM OF SPECIAL TYPES OF INJURIES

Incised, piercing, and flap wounds are produced by sharp instruments or objects applied with more or less force; while contused, lacerated, and bite wounds are more often of the nature of tears produced by blunt instruments.

Small objects, as motes may be borne by the wind, carried to the eyes, stick to the conjunctiva, and become impacted therein, or are carried under the lids and by rubbing may be impacted into the cornea. Others come with force from the breaking of an object and are impacted directly, or, if hot, burn themselves a place in the tissues.

Explosions of firearms, powder and dynamite result in the impaction of foreign bodies, not only from the exploding compound, but also from surrounding objects.

Contusions cause both direct and indirect damage; the direct by impaction of the object; the indirect from rebound of the tissues from the elastic contents of the orbit and its bony walls. Ruptures occur through compression and inward pressure, which, when the coefficient of resistance of the structure is reached, causes disassociation of contiguity and bursting. If the structure gives under the force, it breaks away from its surroundings and direct rupture with dislocation results.

Rupture of the cornea or sclera is produced by the pressure of the foreign body being so distributed that there is no place for the globe to go and it thus bursts.

In direct rupture the break occurs at the point of pressure in another part of the globe. The mechanisms of corneal, scleral, iridic and chorioidal ruptures are under the anatomic heading.

The mechanism is mostly that of burning, in solar that of burning com-

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bined with the electro-chemic effects of ultraviolet light, in electric injuries burning, light, and electrolysis, and in some instances the effect of concussion.

The effect of explosions of gunpowder and dynamite, and of firearms, causes, through a combination of wounds, with and without penetration of foreign bodies, contusion, concussion, ruptures, burning and infection.

Infected wounds have the characteristics of other wounds, combined with loss of tissue from ulceration due to necrosis, following the development of microörganism within. The infection may be carried by the object producing the injury, by accompanying foreign bodies, exposure to the air, or by contact with fingers, unclean instruments, medicaments, or bandages.

PROPHYLAXIS OF INJURIES OF THE EYE

While many eye accidents are unavoidable, yet the large majority are preventable by due care of the patient, parents, fellow-workmen or employers.

In agricultural life many accidents occur from carelessness, as injuries from baling wire, straw, hooks, branches, splitting wood, horning from cows, kicks from cows, horses, etc., most of which are to be avoided by due care.

Safeguards against accidents to workingmen have been forced upon the attention of manufacturers, transportation companies and others, not only by legal measures, but by the necessity for conservation of their own goods and machinery, the loss of service and the cost of care and expense in treatment of such workingmen, as well as protection from damage suits, which give lawyers lots of work. The policy of making factory work safer and more healthful is profitable as well as humane, and it makes the workman more contented. Safety appliances are in use in most dangerous trades and have markedly decreased the proportion of accidents, particularly of the eye. Note the lessened number of blind from accidents within the last 10 years. But it is with reluctance that the workman uses them and he will shirk their application unless carefully watched and continuously warned. Even if protectors hang alongside of the grindstone they are rarely used. There are a variety of protectors in the market. Gauze wire, fitting close to the eye like a cup and attached to the head by a string, is employed by stone breakers, and in some iron works. The mesh should be sufficiently strong and fine, and sufficiently close to prevent, as far as possible, even small chippings passing through it, and yet to interfere with sight as little as possible.

Large heavy glasses made of plain glass with heavy frames are used by stone masons; plain glass with leather fittings for workers in chemicals and in lime works where the acrid fumes may affect the eyes. Automobile protectors are now in common use for out-of-door people using these vehicles.

Amber, novial, Crookes' and even plain white glass not only offer protection from small flying objects, but in a great measure cut off the violet or chemical rays.

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Where grinders or workmen in the iron trades sharpen their tools a number of times a day at the wheel, thousands of foreign bodies fly about, many of which strike the person despite safety appliances that may be used. I have in my possession a pair of ordinary spectacles used by a workman, which is completely studded with hundreds of abrasions caused by flying particles of emery—a not uncommon observation.

If one examine carefully the cornea in the eye of a grinder he may be surprised to find a large number of specks, which are the results of previous in-

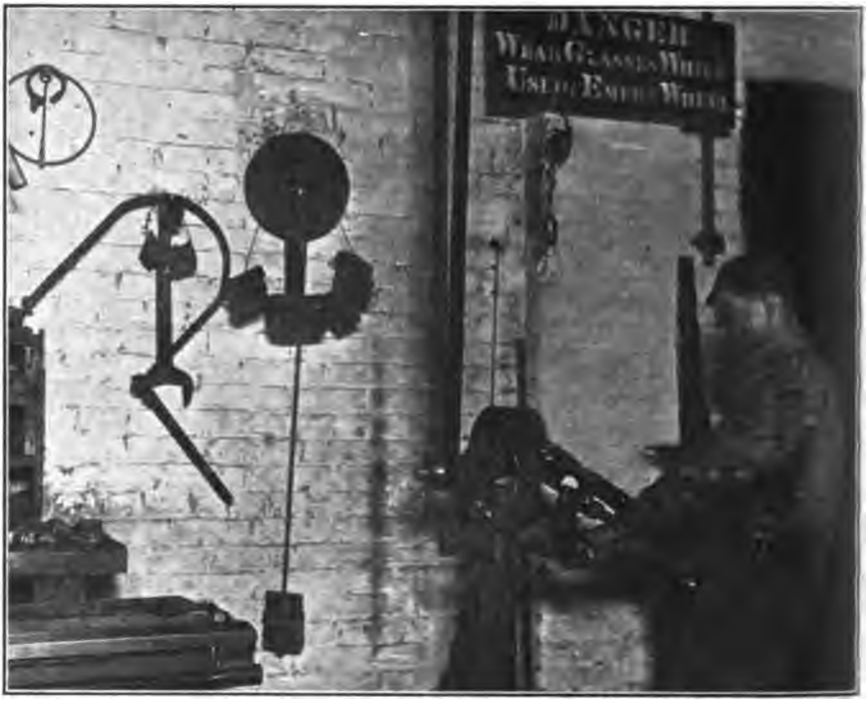

FIG. 15.—Men at emery wheel should at all times wear goggles even if wheel has a permanent glass guard. U. S. Steel Corp'n. (*Würdemann.*)

juries. If they be situated without the visual area they give rise to no inconvenience, but if in the visual axis, a very small scar may cause decided lowering of the visual acuity. All such trivial accidents could be prevented by the workingman wearing protection while at such forms of work.

The use of the pneumatic chipper has, in many cases, almost superseded the hand chipping by sate and hammer, and thus the dangers of chipping are largely avoided. It accomplishes in 1 hour what would take 6 hours by hand, and prevents the flying about of fragments, as it is more like running a scoop over fairly soft cheese than using a tool on hard steel.

The proper arrangement of men at their work and the use of screens

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to avoid injury to their fellow-workmen and to passers-by have, together with the wearing of proper eye protectors, conserved the safety of sight in factories where such have been installed and made obligatory.

My experience has shown that there is less difficulty in enlisting the support of the employers than the assent of the men to adopt precautionary measures.

PROTECTIVE LEGISLATION

Our own workmen are protected by various laws for the regulation of machinery, of different natures in the several states, and in the case of public carriers, as the railway and steamship companies, by the Interstate Commerce Commission and by their own stringent rules, which, however, have more to do with conservation of the public safety and their own goods and machinery than with the individual workman.

Study of the appalling list of accidents occurring annually in American industries has led employers to install many modern devices for making factory work safer and more healthful. The policy of doing this is profitable as well as humane, for it makes workmen brisker and more contented.

Devices have been put into use, moreover, to prevent accidents, and provision has been made to attend to the men injured when accidents occur. Ten years ago an accident would drive a factory engineer away from his throttle at the critical moment. Nowadays a factory girl can shut down the most ponderous engine by pressing a button. Accidents do happen, however, many of them. In the best regulated factories the victim goes to the factory hospital.

In case of negligence being shown on the part of the employer by reason of defective tools, surroundings, or placing the men at extra dangerous work for which they have not specially contracted and from which circumstances accidents arise, the workmen are entitled by law to recover some compensation. In all such suits, however, the compensation asked for is more, and ofttimes many times the amount, than should be allowed. We claim that all compensation, not alone that to be given as the result of claims at law but also claims for insurance, should be regulated by the amount of the economic damage.

By the term "workmen's compensation laws" is meant enactments which embody the principle that the workman is entitled to compensation for injuries received in the course of his employment. Such laws have been enacted in 31 states of the American Union, Alaska, Hawaii and the Panama Zone, but are far from uniform, showing the widest diversities in the methods and amounts of compensation and scope of the laws.

Usually the injuries must cause disablement for a specified number of days or weeks before compensation becomes due. The employer may usually be relieved from the payment of compensation if he can prove that the injury was caused intentionally or by willful misconduct, or, in some

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countries, by the gross negligence of the injured person or during the performance of an illegal act.

The industries usually covered by the acts occur in manufacturing, mining and quarrying, transportation, building and engineering work, and other employments involving more or less hazard. In Belgium, France and Great Britain the laws apply to practically all employments. In Austria, Norway, Russia, Spain and Sweden only workmen engaged in actual manual work, and in some cases those exposed to the same risks, such as overseers and technical experts, come within the operations of the law. On the other hand, in France, Great Britain, the British colonies, and Hungary the laws apply to salaried employees and workmen equally. Overseers and technical experts earning more than a prescribed amount are excluded in Belgium, Denmark, Germany, Great Britain, Italy, Luxemberg and Russia. Employees of the state, provincial and local administrations usually come within the provisions of the act.

The entire burden rests upon the employer in all but four countries—Austria, Germany, Hungary and Luxemberg, where the employees bear part of the expense. The laws in every case fix the compensation to be paid. Except in Sweden the compensation is based upon the wages of the injured person. It consists of medical and surgical treatment and periodical allowances for temporary disability, and annual pensions or lump sum payments for permanent disability or death.

In America and in other countries the estimation of the damage done by accidents in legal cases is left to the empirical dictum of the judge, who is guided by precedent, and to the sympathies or prejudices of the jury, which may be aroused by the attorney presenting the case, pain of body and anguish of mind being apparently very important factors.

Diagnosis of Injuries to the Eye.—The diagnosis of injuries to the eye is made by subjective and objective examinations, of which the history, symptomatology, and the visual acuity, field, condition of the ocular musculature and the refraction are given their proper consideration.

Objective examination by direct and focal illumination, the ophthalmoscope, diaphanoscope, sideroscope, magnetic attraction and the X-ray offer exact means of diagnosis.

A careful history should be obtained, giving the date and hour of the accident, the character of the work and surroundings, the instrument concerned, object or foreign body causing the lesion, and, in special cases, the names of the witnesses to the accident; also the character of attempts to remove a foreign body, and whether or not the case has been attended by a physician, are facts that should be elicited. Remember that, while the interests of the patient are of prime importance, we should be guarded in our prognosis, as it depends very largely upon previous injuries and the character of the first dressing. We should never give an opinion reflecting upon the first consultant, or as to the liability of the employer, for these are theories

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that the lawyers may be allowed to fight about and the courts decide. The less we have to do with the legalized human parasites who are in the habit of soliciting personal damage suits, the better it is for the physician, and we should not furnish information in advance upon which a personal damage or malpractice suit might be based.

Usually an examination may be conducted without a local anæsthetic, as cocaine or holocaine, but such are frequently needed to subdue irritability, adrenalin to lessen congestion, and cocaine, homatropine, euphthalmine, or atropine for mydriasis. The visual acuity, and in some cases the visual field, muscle balance, duction and versions, the refraction, etc., should be ascertained. In fact, in medico-legal cases a full examination should always be made, and sketch drawings or water colors may be made, as well as full notes taken. The literature of the subject should be looked up, as then the examiner, as a medical witness, will be fully prepared for his answers in court. The demeanor, willingness of the patient and actions of his companions should be noted as thereby hints as to malingering may be ascertained.

The examination of an injured person from a medico-legal standpoint should be conducted in a sympathetic manner in order to obtain the patient's confidence, and while the examiner should ever be on the watch for malingering, yet he should not take an antagonistic stand. Such an examination is often grudgingly submitted to, the patient looking upon the examiner in the light of an opponent who has been hired by the defense to find some excuses for prevention of the collection of damages.

The opposite frame of mind is shown by those injured persons who themselves secure an examination as a basis for malpractice, or personal injury suit. Yet they, too, invariably distort and magnify the importance of the economic damage.

It behooves us, therefore, in examination of injury cases, to leave no impression upon the patient as to the advisability of his seeking recourse at law, but to conduct the examination in an impartial and sympathetic manner, at the same time leaving a favorable opinion upon his mind.

A comparatively small proportion of accidental injuries have, in American law, a basis for collection of damages; the larger number have been worked up for pecuniary rather than eleemosynary purposes. Be this as it may, one should not lose sight of the fact that cases occur in which there are really grounds for damages and which should obtain pecuniary relief thereby.

Owing to the transparency, translucency, and delicacy of the ocular structures a number of special methods of examination are of help, of which general inspection by direct and reflected light should first be used.

Objective Examination.—Inspection by direct illumination by daylight, electric light or reflecting mirror is first made. The skin, lids, cornea, conjunctiva, puncta, lacrimalia, etc., are observed. Then the retrobulbar folds and undersurface of the lids are brought into view by eversion with the fingers, but preferably by pushing down the retrotarsal folds by a smooth

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instrument, as a small glass rod, handle of an instrument, or cotton-tipped stick, as it is here that most diseases of the conjunctiva are prominent and foreign bodies may be impacted therein.

Many small and otherwise almost indistinguishable abrasions of the corneal epithelium, wounds and small foreign bodies in the cornea, may be brought into view by the staining of the tissues with a 2 per cent. fluorescein

FIG. 16.—A dangerous practice which should be absolutely forbidden, and which causes much infection. When injured report at once to the emergency hospital. U. S. Steel Corp'n. (*Würdemann.*)

and 2 per cent. bicarbonate of soda solution. This aniline dye will not stain the intact corneal epithelium, but readily passes into the subjacent parenchyma and abraded epithelial cells, forming a bright green background upon which foreign bodies are readily perceived.

Magnification of the eye by a lens, preferably by the Berger or Jackson binocular loupe, is of great value.

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Focal illumination in the dark room is ordinarily carried out by focusing the light upon the eye by a large loupe. The use of the diaphanoscope for this purpose gives, however, a much better illumination, as the light is confined to a narrow beam. The author's transilluminator is the size of an ordinary fountain pen and is as readily handled.

The ophthalmoscope should first be used at a distance of a couple of feet from the eye and magnification obtained by use of a +3.00 to +16.00 lens in the instrument, the patient being directed to look in various directions, whereby a foreign body may be brought into view against the red background of the fundus; approaching closer to the eyes, after the cornea and anterior chamber have been examined in this manner, the lens, vitreous, and fundus are then successfully investigated. The electric ophthalmoscope with light regulated by a rheostat gives the most elastic means of such examination.

Diaphanoscopy is a means of rendering the eyeball luminous, whereby the shadow of the ciliary body, iris, tumor, or large foreign body within the eye may be observed. I have a number of times determined the presence of foreign bodies which were at first indiscernable by other methods behind the iris, in the lens or in the fundus by this method.

The sideroscope, if properly made and mounted, will determine the presence of a foreign body, but it must be magnetic, and the accuracy with which you are able to locate foreign bodies within the eye often varies from 1 to 3 cm.

"The instrument is a marvel of delicacy, but to handle it requires the patience of a saint."

The giant magnet will reveal the presence of magnetizable pieces of steel and iron by causing traction within the eye, varying from a drawing sensation to decided pain on approach of the eye to the magnet. The foreign body may likewise be drawn into view, or the eye may adhere or curve forward toward the magnet if the body be sufficiently large. Such a method of examination is, however, somewhat dangerous, on account of the foreign body, while approaching the magnet, acting as a missile and tearing structures that might not have been previously injured. In such examination the eye, needless to say, should not be anæsthetized.

The increasing use of magnesium and nickel steel renders the number of non-magnetizable foreign bodies somewhat larger in proportion than previously.

The exact determination of the existence, position and size of foreign bodies within the globe has only become possible since the advent of the Röntgen rays.

As the frequency of injuries to the eye from flying copper increases in number each year, due to more extended use of that metal, largely from the increased use of electricity, the value of accurate radiographs becomes more apparent.

In a series of experiments to determine the degree of penetration of the X-rays to various substances, such as glass, marble, granite, cement, etc.,

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Sweet placed particles of these substances in the inner canthus of a cocanized eye. The size of each was approximately 2 × 2½ mm. The result shows that all of these substances can be made visible if proper technique be followed. The figures show the density of shadows of each materials, the exposure being the same for all materials, coal being the only one used that failed to demonstrate a shadow of any usefulness.

Accidental injuries to the eyes may be imitated by the patient putting in medicinal or irritative substances or wounding or otherwise causing actual injuries to his own eyes (self-inflicted damage), or, what is more commonly the case, alleging loss of sight or other function or liability to use the eyes, or painful affections (simulation), or overstating the degree of damage to the function of sight (aggravation)—all of which classes under the generic term of malingering.

More difficult to determine are those cases where the existence of accidental lesion is apparent in which the injured party claims greater functional damages than actually exists in the individual. A number of observations should here be made in order to determine the proper relations.

THE RELATION BETWEEN ACCIDENTAL INJURIES AND PREVIOUSLY INTERCURRENT AND POST-TRAUMATIC CHANGES IN THE EYE

Conditions due to an accidental injury must be differentiated from those existing before, those concurrent with, or those occurring after the injury from other causes.

In uncomplicated fresh cases, blemishes due to previously existing disease, or the results of former accidents, are readily determined, but especially those which come to the examiner a long time after the alleged injury; such may not be so evident, and afford weighty problems.

It is especially necessary in these cases to make a general examination of the body and to carefully note the condition in the non-injured eye. In the injured organ note whether typical scars or inflammatory changes or their effects are to be seen.

Many persons suddenly discover that they cannot see well with one or both eyes after an accident, whereas from myopia, high astigmia, hyperopia, or even presbyopia they have previously been poor sighted and have not known of the condition. Some are honest in their claims; other seize the opportunity afforded by an accident to blame the deficiency upon the alleged trauma.

THE RESPONSIBILITY OF THE PHYSICIAN

Legally, the physician may be said to have no responsibility, as regards prognosis, and this is a matter of personal opinion rather than of fact, but ethically his position is one which is twofold. From his own standpoint and

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that of the patient's good it is necessary that the patient retain a favorable opinion of the physician and, in many cases, continue to take treatment. Thus, by an unfavorable prognosis which may not prove to be true—for wonderful are the ways of Nature in curing disease and healing injury—the physician may be discredited by a partial or complete restoration to health of the patient. Pecuniarily he may suffer as a result of the patient leaving him to go to some other physician who gives a more favorable prognosis. Secondarily the effect upon the patient's mind is one that should be considered, and hope should always be afforded to what is esteemed the hopeless cases. While the whole truth should be told the family physician and to the family of the patient, yet the whole, and what may be most unpalatable, truth need not be told the patient himself. This has to be learned by experience in the most unpleasant manner by the average physician as the result of an unfavorable prognosis.

Prognosis of Injuries to the Eye.—The prognosis of ocular injuries depends largely upon the part of the eye that is damaged, more especially that having to do with the function of sight. Of first importance is the central visual acuity; secondly, the visual field; thirdly, the ocular movements; and lastly, the light and color sense. To these may be added the cosmetic damage and the ability to use the eyes for work, or, as Magnus puts it, the ability to compete; and the sum of them all results in the economic vision.

Trauma acting on the parts of the eye necessary for clear vision, as the visual zone of the cornea, the lens, vitreous, macula lutea, does more damage in proportion than that to one side, and offers a more unfavorable prognosis on account of resultant obstruction to vision. Injuries to the optic nerve and visual sphere are usually followed by atrophy and blindness. Injuries of the ciliary region are provocative of sympathetic ophthalmitis and loss of the other eye as well. As a rule, clean-cut wounds heal well if not infected. Infection may, as a rule, be successfully combated if seen in the early stages. Retained foreign bodies are always dangerous. Contusions are generally dangerous, as they lead to secondary degenerative changes and detachment of the retina. Burns of the anterior portion of the globe are always to be feared, lime injuries especially. Electrical injuries result in primary or secondary damage to the lens and retina. Double perforating wounds of the globe, even with retained foreign bodies behind in the orbit, are compatible with comparatively small amount of damage to the function.

Local fractures of the orbital walls are of less moment, *quoad vitam*, than those which extend along the base of the brain.

The future prospects for eyes that have been injured opens a wide and doubtful field for prognosis, but as the physician is frequently asked in court concerning prognosis, and as this is a matter of constant inquiry from patients, he must have certain knowledge of the subject and be prepared with proper answers. While upon questions of prognosis it is not well to make definite statements directly to the patient—on account of the psychic element, with

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dangers of loss of the patient or his suicide from unfavorable prognosis—the family physician and the family should be made fully conversant. There are certain lesions of the eye which almost invariably result in loss of function and even in loss of the eyeball itself; for instance, ulcerations of the cornea are as a rule followed by opacities, varying from nebulæ to leucomata, which materially diminish the visual acuity, especially if these be in the visual axis. Likewise, lesions complicated with iritis may, if not properly treated, result in occlusion of the pupil. Injuries of the lens almost invariably result in total opacity, although there have been a number of cases observed and reported in which foreign bodies have remained *in situ* for a number of years without causing further opacity; and development of partial cataract is observed in many cases. To this we refer to cataract from electricity and distinguish true contusion of the lens.

Contusions and wounds of the ciliary region frequently result in chronic irido-cyclitis and atrophy of the bulb. Retained foreign bodies in the eye result in blindness from such causes, as well as from sympathetic ophthalmitis.

Many cases of amaurosis follow, as is well known, a blow on the head, of which the pathogeny is an indirect fracture through the optic canal, or an effusion of blood in the nerve sheath. But more frequently is it the case that, following a trauma of the cranium, the injured person, in good faith or not, claims the visual loss to be due to the injury, while the lessened vision may have existed prior to the accident. Of course, when the case is seen just after the trauma and amaurosis exists it is impossible, before some days have elapsed, to discover any optic nerve change. Optic nerve atrophy will follow in a short time. The optic papilla will guide in these cases. When the case is seen long after the accident, while the etiology is determined with less certainty, nevertheless to the trauma of the cranium may be attributed the cause if the contour of the atrophied papilla is clear.

Precaution and attention to apparently trivial details of treatment would curtail in a marked degree the number and success of personal injury suits.

CONSERVATION OF THE WOUNDED EYE

The handling of an injured eye should be conservative from the first, to keep vision, preserve the appearance, relieve the agony, and even in some cases to save life.

The public should be taught that the best first aid to injured eyes is, as a rule, to let them alone, apply clean cloth bandage and to immediately seek a physician, more especially an oculist, except where large quantities of foreign material as sand, dirt or corrosive substances, as lime or chemicals, enter the eye; then the first application is free douching with clean, clear water.

The radical treatment of injuries to the eye is practically summed up in the word enucleation, nearly all other procedures being conservative.

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The therapy of the wounded eye is based upon general surgical principles, and from beginning to end the watchword is asepsis, or cleanliness.

Concerning the septicity of the wound, all chips of metal entering an eye may be considered as free from organisms, the septic inflammations being produced by the subsequent entry of organisms either through the tract of the wound or during the operation for removal.

It is universally recognized that when the foreign body is in the anterior segment of the eye, the injury is much less serious than when it has passed on through the lens into the posterior segment or has entered the vitreous chamber through the ciliary region or the sclera.

A different story of the dangers, damages and results must be told about foreign bodies in the posterior segment of the eye, for as a rule these have flown with much force, have already wounded the anterior structure in their course, and if allowed to remain in the eye not only destroy the sight and cause atrophy of the globe, but may result in sympathetic ophthalmitis.

For the technical treatment of injuries to the eye the reader is referred to the text-books on ocular therapeutics.

II. DISEASES DUE TO OCCUPATION INCURRING EXCESSIVE EYE STRAIN

The second group consists of neuropathies, as nystagmus of miners, spasms of the orbicularis of watchmakers, of the ocular muscles from military drilling, and to the production of eye strain from the close study of students and in occupations requiring prolonged use of the eyes.

But few oculists see the typical nystagmus of miners, but to those who live in coal-mining districts as well as to the workmen and employers this subject is of great importance. This is due to the constrained position of the workers, either in excavating the coal or in inspectors from tapping the roof to see if it is safe.

The movements of the eyes are mostly rotatory, occasionally to and fro, but seldom vertical. The oscillations may be stopped by turning the eyes downward. Five to 20 per cent. of miners suffer and have to quit the occupation for intervals or altogether.

Nystagmus is also met in engravers, draughtsmen and compositors, jewelers and typists, but is only incidental. It is similar in type to writers' cramp and is speedily relieved by rest.

Now we come to the great subject of eye strain from muscular and refractive defects, to which perhaps 90 per cent. of civilized eyes are subject, and which is brought on by close use of the eyes and is relieved by lenses and ocular muscle operations, and not to be elaborated here as thousands of pages of print on this subject may be found in text-books and medical magazines. It must be remembered that perfectly normal as well as diseased eyes may suffer from glare and eye strain from overwork.

The production of myopia in schools and by close work is a live issue.

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Certain races as the Teutons and the Jews seem more apt to develop this anomaly, which in some leads to grave changes in the eyes and loss of visual acuity or even blindness; for this see other text-books.

III. DISEASES DUE TO EXCESSIVE EXPOSURE TO LIGHT AND HEAT

The constant gazing at bright objects, as by dentists in gold filling and by electroplaters, may cause evanescent scotomata which, though alarming at the time, disappear on resting and may be prevented by the use of appropriately tinted glasses.

A more severe affection may be seen in steel melters, electro-welders, glass workers and furnace men and in exposure to strong light, when the brighter light may not only temporarily use up the visual purple but cause a degeneration of the retina at the macula similar in type to that caused by looking at the sun in a solar eclipse. Cobalt and dark glasses may prevent the occurrence.

The heat of melted iron, glass, electric and other furnaces, etc., is responsible for burning of the lids, cornea and perhaps even for the cataract of glass blowers and degeneration of the retina. Protection by dark glasses and helmets prevents this occurrence.

Short circuiting of electric wires give rise to terrible general burns, and the electrolysis to formation of cataract and changes in the retina, amblyopia and blindness. Most of these accidents are due to carelessness of employees and are to be prevented only by education and intelligence.

IV. TOXICOLOGY—SYSTEMIC POISONING CAUSING LOSS OF SIGHT

The ingestion, inhalation, epidermic, hypodermic, endermic, intramuscular or any other method by which certain toxic substances may get into, or be developed within, the system induces changes in the tissues of the eye by which diverse effects are produced, denominated by the term toxic amblyopia.

Among the commonest poisons thus affecting the eye are alcohol, tobacco, iodoform, naphthalin, caffeine, acetanilide, salicylic acid, atoxyl, arsenic, felix mas, santonin, and pomegranate, to which some persons have an idiosyncrasy, particularly as regards damage to vision and the eyes, even in medicinal doses.

Arsenic is extensively used in wall paper, clothing, artificial flowers, making Paris green and in paints, and these workers, as well as those who take it medicinally or use cosmetics, are liable to suffer from eczema of lids and conjunctival hyperæmia as well as in a few cases from amblyopia and optic neurits.

Of those occurring in trades, there have been reported cases of miners'

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arsenical poisoning with resultant optic nerve atrophy, following the dust from the brittle mineral known as sulfarsenide of cobalt; but as arsenic is usually found in the form of arsenical pyrites it does not seem to cause any injurious effects.

Likewise in ordinary lead mines *lead poisoning* is almost unknown, as it is usually mined in the form of galena, the sulphate of lead, a heavy and quite insoluble substance. In carbonate mines, the earthy lead carbonates give rise to lead poisoning, and in a few cases paralysis of the extrinsic muscles of the eye has been noted. In the production of the white oxides of lead for paint, plumbism has been often seen, and with the general symptoms paralysis of the ocular muscles has been noted. Lead is used in many occupations, especially painting, plumbing, file cutting. No acute eye cases are reported, but chronic cases from inhalation and wetting of fingers, holding of paint brushes and tools in the mouth, central and peripheral contractions of the visual field are seen from retrobulbar neuritis; palsy of the eye muscles is also encountered.

Amyl alcohol or *fusel oil*, found in impure wines, whiskies, etc., seldom causes complete blindness and is rarely known in trades, generally following chronic alcoholism usually associated with tobacco.

It may be that cases have occurred of *tobacco blindness* in producers and handlers of tobacco from inhalation of the dust or from licking the leaves as is sometimes the filthy habit of cigar makers, but I have never seen such. Those tobacco workers that I have encountered with toxic amblyopia were all heavy drinkers.

Methyl alcohol or *wood spirit* is frequently used in the preparation of varnish, lacquer, polish, perfumes and other industries enumerated in the list of industrial poisons (see page 731). Cases of conjunctivitis, also serious affections of the retina and the optic nerve resulting even in blindness from atrophy of this nerve, have been reported.

Tea tasters consume an enormous amount of the tea and I have seen several cases of amblyopia in these people, who have all recovered when they have taken a vacation from their vocation together with appropriate treatment.

Bisulphide of *carbon* in rubber making has been shown to produce amblyopia. It is less used than formerly in curing rubber.

In the manufacture of *explosives*, dinitrobenzol is responsible for general changes in the blood and nervous system and in the making as well as the using of same for explosive charges, especially in mines, has given rise to toxic amblyopia.

In dye factories and in the manufacture of coal-tar products there are complaints of irritation of the eyes and loss of vision from its inhalation and absorption, possibly due to *aniline* poison.

Iodoform is used in surgery and has caused kidney and eye lesions, but no evil effect has been reported from its manufacture.

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THE ESTIMATION OF ECONOMIC DAMAGE

Upon the use of the organ of vision depends the earning powers for the large majority of trades and professions; thus earning ability is economically synonymous with visual earning ability.

It is self-evident that a totally blind person is absolutely incompetent in any trade or profession requiring eyesight. An economically blind person—*i.e.*, one whose visual acuity is less than 5 per cent. of the normal—is in the same position, for, although he may be able to get about, the vision is not sufficient to allow of even the lowest grade of remunerative work. The vast majority of blind people are not only incapable of earning anything, but are a charge upon their families and upon the community. It is true that there are certain exceptions to the above proposition. There are, and have been, blind persons who have become poets, machinists, chair makers, broom makers, etc., but cases that have become economic factors are so unusual as to be commented upon in the public press and held up as especially talented and well-placed persons who, by great labor of their teachers, and by their own exceptional diligence, have been so highly educated as to be able to meet, in a measure, with the competition of normal individuals. In the case of an adult suddenly becoming blind, his previous economic education goes for naught, and he at once steps out of the ranks of workers.

Nearly all trades and professions require good eyesight, even the coarsest sort of labor being affected if the vision falls below 50 per cent., and being impossible if it is below 0.05 per cent. of the normal visual acuity. For finer kinds of work, the visual range is between 75 per cent. and 15 per cent. A workingman who either suddenly or gradually becomes blind loses his job, and with it his earning ability. Aside from the loss of time and wages ensuant upon the injury and convalescence therefrom, poor sight certainly affects the amount and character of work, the quality and output diminishing in a direct ratio to the loss of sight, until a degree is reached where the person cannot work any more. The remuneration for work necessarily depends upon its amount and its quality. Thus injury to vision generally necessitates loss of earning powers.

The pecuniary value of a man's life may, for our purposes, be expressed by the amount of money that he may earn in the course of his life. We stated above that the visual earning ability was economically synonymous with the full earning ability, and we may thus value vision with the pecuniary valuation of life.

We may say that "sight is priceless" and "vision is not a commodity that may be purchased or disposed of in the market," for there are few persons who would voluntarily allow of the infliction of any unnecessary bodily injury for any compensation whatever; but such matters of ethics do not fall within the pale of our present discussion. We are dealing with the established economic fact that injury to vision of more than a certain extent neces-

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sitates limitation of the amount and character of work. Following upon this, it is easily deduced that the amount of wages received would be less.

The question now arises, how are we to reckon the loss of wages? This we may do from experience in examining large numbers of individuals. It has been found that the loss of vision of a certain amount results in a certain effect upon the earning ability of the individual. We may also deal with futurities, and figure the probable loss in any given case by finding the percentage of damage to the normal function, and apply our reasoning to the calculation of the probable pecuniary loss. In order to do this we have accepted the visual earning power as equivalent to the total earning power.

The Formula of Magnus.—Magnus therefore evolved a formula which we have accepted as the simplest and most appropriate, which considers the visual earning ability as composed of the several factors entering onto vision together with the ability to compete, expressed as an arithmetic equation.

Thus:

E $= F\sqrt[2]{K}$.
E = Earning ability = ocular earning ability.
F = Visual act.
$\sqrt[2]{K}$ = Ability to compete.
F $= C$ (maximum)$\sqrt{P}\sqrt[2]{M}$ = physiologic act of vision in which
C (maximum) = maximum acuity of the better eye.
$\sqrt{P}$ = Binocular visual field.
$\sqrt[2]{M}$ $= \sqrt[4]{M}$ = normal value of ocular muscles.

This is modified by the ability to compete:

$$\sqrt[2]{K} = \sqrt[2]{\frac{C_1 + C_2}{2}\sqrt{P}\sqrt[4]{M}}$$

in which $\frac{C_1 + C_2}{2}$ = the added acuities of both eyes, $\sqrt{P}$ the visual field, and $\sqrt[4]{M}$ the muscular action, the complete formula for the ocular earning ability being

$$E = C \text{ (maximum) } \sqrt{P}\sqrt{M}\sqrt[2]{\frac{C_1 + C_2}{2}\sqrt{P}\sqrt[4]{M}}$$

which is the mathematical expression of the earning ability and which may be readily calculated by anyone who can do a simple algebraic equation, as it is reduced to a simple multiplication example by using tables which have been compiled for the purpose, as the algebraic signs are replaced by figures which are readily found therein. The roots are readily calculated or may be found in our tables. Würdemann has added monetary computation to this formula and the method is now known and accepted as a scientific fact and is received as such in present forensic practice. (Visual Economics, Magnus and Würdemann, 1902, and subsequent essays.)

Résumé.—1. The former usages for the estimation of pensions, insurance,

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and damages at law, from injury to vision, are based wholly upon precedent and are purely empirical.

2. The relation of the visual act to the earning ability is susceptible of mathematic demonstration.

3. The probable loss of wages—*i.e.*, the effect on the earning ability of the individual—may be determined by the particular injury to vision.

4. (*a*) Insurance contracts will probably be continued under the present business arrangements, but may be made equitable, subject to the amount of economic damage, a percentage of the sum for total disability being paid for partial losses. In the case of loss of vision of one eye the rates should be modified to between 18 and 30 per cent. of the total disability.

(*b*) For the settlement of pensions and annuities the full annual economic damage should be paid.

(*c*) For the settlement of claims at law the probable economic damage should be estimated and considered the principal element, subject to business discount, to additions for the actual expenses consequent on the accident and empirical amounts for the pain and anguish thereto incurred, contributory negligence and other legal factors being also considered in the verdict.

5. The calculations and rules of Magnus and Würdemann afford a method of estimating the amount of the probable economic damage in a manner fair and just to all parties and agreeable to all legal demands.

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CHAPTER II

GLASS-WORKERS' CATARACT

BY T. M. LEGGE, M. D., London, England.

Following on the Workmen's Compensation Act, 1906 which, in section 8, applied the principle of compensation as in the case of accidents to poisoning by lead, arsenic, phosphorus, and mercury, and to the two infections by anthrax and ankylostomiasis, a Committee was appointed to consider what other diseases were due to industrial occupation, were distinguishable as such, and could properly be brought under the provisions of the section. One of the most interesting diseases thus brought under review, and the one upon which there was the greatest difficulty in coming to a decision, was occurrence of cataract in glass workers. In regard to the disease four questions had to be decided: (1) Did it exist? (2) Was it much more frequent in glass workers than in the general male population? (3) Was it characteristic? (4) Would it be in the interests of the workers themselves to schedule it since not the fact of having cataract entitled to compensation but only incapacity resulting from it?

The attention of the Committee was first directed to the subject by the evidence of the secretary of the glass-bottle makers of Yorkshire United Trade Protection Society—a signal instance of the value of the material contained in such Society records if only they are properly used. He suffered from defective eyesight himself and in noting the sickness claims was struck by the frequency with which cataract appeared as a cause leading to superannuation. Thus he was able to show that among some 2000 members of whom 114 were superannuated, no less than 34 (30.0 per cent.) had suffered or were suffering from cataract. In another similar trade society with 1000 members it was ascertained that 6 out of 30 members on the superannuation fund were disabled on account of cataract.

The employers, on the other hand, attached small importance to the ailment no doubt because the number of cases in proportion to the number of men employed was small and, in individual works, so extremely small as to make some of them deny its existence as a trade disease. The matter, from the administrative point of view, was further seriously complicated by the fact that employers threatened in self-defence, if glass workers' cataract werc scheduled, to have periodical examination of the eyes of the persons employed made and to discharge, many years it might be before there was any incapacity, those showing changes in the lens. Nor did the Committee at the time they had to report know of any statistics showing what the incidence of cataract was among the general population. If cataract, they said,

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was found to be 10 times as frequent in glass workers as among other people of similar age the disease ought to be scheduled, even although in one case out of ten the employer would be required to pay compensation to a man in whom it would have developed had he never been employed in that trade. The matter, therefore, was left over for further inquiry which I was asked to make.

Already, in 1903, Dr. W. Robinson, Surgeon to the Sunderland and Durham County Eye Infirmary—a district comprising several bottle and pressed glass works—had published a paper in which he concluded that hard cataract in bottle finishers was very common. "How much commoner it is than among the rest of the community is shown by the fact that 18 out of 75 (1 in 4) hard cataracts operated on last year in our Eye Infirmary were in bottle finishers, there being only 200 or 300 bottle finishers in a population of nearly a million and a quarter in this county."

Briefly described the work in a glass-bottle factory is as follows: The mixed materials spoken of as "metal" and consisting of sand, clay, lime, marl, etc., are thrown by the "founder" into a furnace or "tank" kept at a white heat of about 1500°C. At the opposite end of the furnace are several openings at which the bottle makers work in squads of five each. The "gatherer" dips the iron blowpipe into the metal, having in doing so naturally to look into the white mass, and takes up sufficient to make a bottle. This he hands to the bottle blower who places it in a mould and blows down the pipe to form the bottle. From the glass blower the bottle, still on the end of the pipe, is handed to the finisher who cuts it off from the pipe. The finisher, standing right up against the "gloriole," looks through this into the molten metal and takes up sufficient to form the rim on the neck of the bottle. He then seats himself, sheltered by a brick projection, and puts the rim on round the neck.

Robinson calculated that during each shift of 11 hours from 110 to 120 bottles are finished, and reckoning the time of looking into the furnace at about 3 seconds a time, the eyes of the finisher are exposed to the glare for 5½ hours a week.

In the making of pressed and flint glass the heat is not so intense and the finisher of flint glass bottles sits some yards away from the furnace into which he need never look. The processes in the manufacture of sheet and plate glass are essentially the same. Sheet glass, however, in the present day is blown not by the mouth but by means of compressed air into huge cylinders which are first annealed and then cut into two halves and flattened out in a furnace kept at a dull red heat only.

As to the character of the cataract, Robinson pointed out in proof of its industrial origin from exposure to the intense heat and light of the furnace that it commenced always at the posterior pole of the lens involving the cortex. Ordinary senile cataract on the other hand developes usually in the form of sectors or radii from the periphery of the lens. His explanation of its origin

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was that the posterior pole was practically the optical center where the principal rays falling on the lens cross and pass on without refraction, and further, the point where undue crowding together of all the rays from the furnace, except the direct and principal, rays which undergo refraction at the anterior surface of the lens.

In a subsequent paper in 1907 Robinson adduced as further evidence of his contention the results of his examination of the men in three glass works in the Sunderland district where among 400 men he found 37 finishers and 3 gatherers who were suffering from cataract.

TABLE 1.—CHANGES IN THE LENS AMONG 513 MEN ENGAGED IN THE GATHERING, BLOWING, FINISHING, AND FLATTENING OF GLASS, AND 278 OTHER PERSONS, ALL 30 YEARS OF AGE AND OVER

Occupation	30–40	Percentage	41–50	Percentage	51–60	Percentage	Over 60	Total	Percentage
Sheet glass—									
Blowers	48	......	10	......	2	......	1	61	......
Positive	5	10.4	2	20.0	.....	33.3	1	8	13.1
Gatherers	90	......	19	......	2	......	.....	111	......
Positive	7	7.8	2	10.5	2	100.0	.....	11	9.9
Flatteners	78	......	23	......	2	......	.....	103	......
Positive	12	15.4	4	17.4	2	100.0	.....	18	17.5
Total	216	......	52	......	6	......	1	275	......
Positive	24	11.1	8	15.4	4	71.4	1	37	13.5
Glass bottles—									
Finishers	88	......	68	......	32	......	4	192	......
Positive	7	7.9	14	20.6	16	52.8	3	40	20.8
Blowers	9	......	1	......	5	......	3	18	......
Positive	......	......	....	......	2	50.0	2	4	22.2
Gatherers	5	......	3	......	1	......	.....	9	......
Positive	2	40.0	1	33.3	....	......	.....	3	33.3
Total	102	......	72	......	38	......	7	219	......
Positive	9	8.8	15	20.8	18	51.1	5	47	21.5
Pressed glass—									
Finishers	7	......	3	......	4	......	1	15	......
Positive	1	14.3	....	......	3	80.0	1	5	33.3
Gatherers	4	......	....	......	....	......	....	4	......
Positive	1	25.0	....	....	....	......	....	1	25.0
Total	11	......	3	......	4	......	1	19	......
Positive	2	18.2	....	......	3	80.0	1	6	31.6
Grand total	329	......	127	......	48	......	9	513	......
Positive	35	10.6	23	18.1	25	56.1	7	90	17.5
Other persons	141	......	90	......	45	......	2	278	......
Positive	3	2.1	8	8.9	8	17.0	.....	19	6.8

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Comparative data, however, were wanting. To obtain them I examined the crystalline lens in the eyes of 513 glass workers engaged in the gathering, blowing, finishing and flattening of glass. Subsequently, under precisely the same conditions, I examined the eyes of 278 persons who were not glass workers.

The results are shown in Table 1.

Viewed in this way changes in the lens are shown to be about five times as frequent in glass workers between 30 and 40 years of age as in other persons, about twice as frequent in those between 41 and 50, and over three times as frequent in those over 50. All classes of workers exposed to incandescent molten glass appeared to suffer, and the differences which are to be noted in the incidence on those making sheet glass, glass bottles, and pressed glass, are not striking. A point of interest was that the class of gatherers in sheet glass works showed considerably the smallest number affected. The heat from the furnace here is so intense that the gatherer is compelled to shield his face with a screen, held in the mouth, with blue glass in front of the eyes.

Mere statement of the number needs to be amplified by character of the changes observed. The very striking differences thus brought out can be tabulated as follows:

Comparison of the Nature of the Changes in the Crystalline Lens of the 513 Glass Workers and 278 Other Persons

	513 glass workers		278 other persons	
	Right	Left	Right	Left
Cataract extracted	3	5	......	.....
Cataract so mature that its nature could not be determined	4	2	......	.....
Posterior cortical cataract	17	19	1	.....
Commencing posterior cortical cataract	5	8	......	1
Dot-like posterior cortical opacity (in central area)	5	2	......	.....
Posterior cortical opacities (not in central area), radii, striæ	4	11	1	1
Peripheral striæ, radii and sectors	14	15	11	9
Other opacities	5	3	1	2
General dulness of lens	7	7	......	.....
Total	64	72	14	13

When viewed with the ophthalmoscope by the indirect method the characteristic change in the lens of the glass worker is opacity of the posterior cortical layers (like a blot of ink) of varying size, within the pupillary area. Changes in the posterior layers were present indeed in over half the positive cases. In the control population examined the characteristic change was, as one would have expected, the appearance of peripheral striæ around the margin of the lens, the central pupillary area remaining perfectly clear. There was exception to this in two cases only and in them the changes could

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not be distinguished from the condition observed in glass workers. The occupation of one of these had been constant attendance for 25 years at an annealing furnace in Woolwich Arsenal—work similar to that of a glass flattener. Among the control population none had sought treatment for cataract, and certainly in not more than three were the opacities so pronounced as to cause any impairment of sight. Among the glass workers, however, in addition to six in whom a single extraction for cataract had been performed and in one a double extraction, there were at least 25 others in whom the sight of one or other of the eyes was seriously impaired by the opacity. Yet it was surprising how almost unimpaired for work the sight appeared to be of several of those in whom small but quite definite posterior cortical cataract was present. Even when vision is affected the bright light given out from the molten glass and the mechanical nature of the work both help to overcome or mask the defect. Were the occupation one necessitating fine work the condition would become apparent much sooner. Many, no doubt, pass through the normal working life ignorant of its existence.

I next sought figures as to cataract as a cause of disablement and superannuation in other trade societies. The Hearts of Oak Benefit Society with a membership of some 300,000 had a large class receiving reduced sick allowance the conditions of receipt of which were that the member should have been on the Society's sick funds for at least 12 months. I believed that if the number of those in receipt of this reduced sick allowance could be ascertained for a number of years fair comparison was possible with the figures supplied to the Committee of superannuated members by various glass-bottle Trade Societies. This I was able to do, finding the incidence of cataract among the 10,549 members in receipt of reduced sick allowance 0.78 per cent. as compared with 22.6 per cent. among the 186 superannuated members of glass-bottle Societies. The average age of incapacity from cataract was in both about the same—56 years. Cataract, indeed, as a cause of disablement among superannuated members of the Yorkshire Bottle Makers' Society considerably exceeded the disablement from every form of heart and lung disease among the members on the reduced sick allowance of the Hearts of Oak Benefit Society.

While the case was thus proved for the industrial origin of the disease the fact remained that it was of very slow growth, and that, during the many years when the efficiency of the workman remained unimpaired, it would be impossible for him to claim compensation. And yet an ophthalmoscopic examination of the eyes would reveal the disease in an incipient stage. The scheduling of the disease without qualification thus opened out grave questions as to the probable effect of such a course upon the prospects of the men affected. These were anxiously considered by the Committee with the result that a half-way course was recommended, namely, the scheduling of the disease but with compensation restricted to cases where an operation was undergone and for a period not exceeding 6 months. Thus not only would

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there be inducement for the sufferer to submit to the comparatively safe operation for the removal of cataract after which usually glass workers are able to obtain employment again—sometimes even at the highly skilled work of finishing, and employers also would not deem it worth the while to incur the expense of arranging for periodical examination or hesitate to employ a glass worker with incipient cataract because of possible compensation to him for a period so short as 6 months.

I was unable to say whether heat rays or light rays caused the condition nor could I suggest an efficient remedy. The combined knowledge of the physicist and physiologist, I said, was required to arrive at a conclusion on these points. This side of the question, however, was taken up by a Committee of the Royal Society among whom were Sir William Crooks, O. M., F. R. S. on the physical side and Dr. J. Franklin Parsons on the physiological.

The former directed his attention to the effect of adding various metallic oxides to the constituents of glass in order to cut off the invisible rays at the ultra-violet and the infra-red ends of the spectrum, *i.e.*, to cut off those rays from highly heated molten glass which are believed to damage the eyes of workmen without, at the same time, obscuring too much light. X-rays were not, he found, emitted by the highly incandescent molten glass. In the radiation from molten glass Crookes found the heat rays in far greater abundance than the ultra-violet, and from this he inferred that it is to the heat rays rather than to the ultra-violet that glass workers' cataract is to be ascribed. It is, however, certain, he adds, that exposure to excess of ultra-violet light also injuriously affects the eye.

By means of extraordinarily exact scientific experiments with apparatus devised by himself and with different glasses made by himself to which known quantities of pure metallic oxides and earthy salts were added, Crookes determined for each glass so prepared:

1. The percentage amount of infra-red rays absorbed.
2. The percentage amount of ultra-violet rays absorbed.
3. The percentage amount of luminous rays transmitted.

To be generally useful a glass should absorb rays of longer wave length than about λ 7200 and so cut off dark heat radiation and it should also be opaque to wave lengths shorter than about λ 3550 thus cutting off the most chemically active rays.

Finally he was able to prepare glasses cutting off over 90 per cent. of heat radiation, opaque to the invisible ultra-violet rays, and sufficiently free from color to be scarcely noticeable when used as spectacles. All three desiderata, however, could not be combined in one and the same specimen. The glasses he describes "include specimens suitable for spectacles adapted to all requirements—from eyes of youth to eyes of age." He gives the composition of the glasses cutting off as much as possible of the heat radiation. Thus in one of a neutral tint cutting off 94 per cent., opaque to ultra-violet rays of shorter

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wave length than λ 3610 and allowing 30 per cent. of incident light to pass through, the composition was raw soda flux 88.5 per cent., black mica 11.5 per cent.; of another enabling the worker to see better at the expense of a little athermancy the proportions were: fused soda flux 88.47 per cent., ferric oxide 1.50 per cent., cobalt sulphate crystallized 0.03 per cent., and cerium nitrate 10.00 per cent. This is of a pale blue and transmits 63 per cent. of luminous rays while cutting off 51 per cent. of the heat rays. Spectacles of this glass, he says, scarcely appear to obstruct light at all and the colors of the objects are practically unchanged.

Selections of glasses prepared according to the formulæ in Sir William Crookes's paper are made by Messrs. Chance Brothers and Co., Ltd., near Birmingham, and at the Whitefriars Glass Works, London, E. C.

Glasses such as these may prevent the incapacity arising from glass-workers' cataract. The disease, however, is so slow in its onset that prejudice against wearing anything interfering in the least with work is great and will have to be overcome. Few glass workers to-day are found attempting to preserve their eyesight by spectacles, partly from the reason stated, and partly because moisture, when perspiration is excessive, condenses on the glass. The latter objection can be overcome by rubbing the glasses with a "Lasin" pencil made of a kind of soap.

Another and very powerful cause is at work which will tend not only to minimize the chance of occurrence of cataract, but also of another occupational disease, which has occasionally been the result of infection from common use of the same blowpipe, namely, syphilis among glass-blowers. To guard against syphilis contracted in this way the French Government has enacted regulations requiring medical examination of glass-blowers at fortnightly intervals. The cause to which I refer is the substitution of machinery for hand-work in the manufacture of glass bottles. A machine is already installed in some works—and the example will be quickly followed elsewhere—which, as it is made to revolve over the margin of the molten glass, sucks up just the necessary amount, then transfers the glass so sucked up to a mould where the bottle is formed by compressed air automatically supplied to it, and finally discharges the bottle in a finished condition ready to be annealed. The machine does its work at the rate of 40 bottles a minute at a cost for labor for the three men needed to supervise its operations about one-ninth that now paid for the same number of bottles manufactured in the old method involving manipulation by 40 men and boys. At present the machine cannot make every kind of bottle. It cannot, for example, introduce the glass marble which forms the stopper in the popular form of lemonade bottle so that for some years to come bottle making by hand must continue. The hard conditions in glass works are just those which should gradually be replaced by machinery and how effectively the new machine achieves this must be seen to be believed.

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CHAPTER III

OCCUPATIONAL INJURIES AND DISEASES OF THE EAR

BY CLARENCE JOHN BLAKE, M. D., Boston, Mass.

Injuries and diseases of the ear, of occupational origin, may be grossly divided into three classes; those which are limited to the external ear, those which affect the sound-transmitting apparatus of the middle ear, and those which are ultimately expended in their deleterious effect upon the more delicate sound-transmitting apparatus of the cochlea and the terminal nerve distributions which constitute the static organ of the labyrinth.

Of these three classes the latter two are more likely to have an intermediate ground of relationship, in so far as major disturbances are concerned, disruptive injuries to the middle ear being sometimes associated, in their consequences, with disturbances of the labyrinthine mechanism and function but labyrinthine disturbances incident to the trades stand apart by themselves, the middle-ear implication being purely secondary to the deeper seated excitation or, possibly, part of a natural protective process. The injuries to the ear, of occupational origin, may range from a solution of continuity of the auricle incident to a blow to the structural changes induced in the membranous cochlea by continued exposure to loud sounds.

Standing outward from the side of the head, to which it is attached by three groups of muscles, in addition to its ligamentous and other soft tissue attachments, composed of a thin plate of cartilage covered with a very vascular skin, the auricle is especially subject to injury in such trades as imply exposure to extremes of temperature, and to possibilities for forcible contact with hard substances.

The effect upon the auricle of exposure to extreme cold in the indoor trades is less frequently observed than that produced by climatic exposure, but the results are the same, a circumscribed dermatitis, sometimes a perichondritis with resultant thickening and, occasionally, a sero-sanguinolent effusion beneath the skin, on the anterior surface of the auricle, with consequent distortion of its contour; the more extreme results of the induced inflammation, necrosis with sloughing and loss of a portion of the auricle, being less frequent in the indoor trades—cold-storage plants for instance—than in cases of climatic exposure, both because the exposure is less and because the subjection to it is more under individual control.

Similar inflammatory changes in the auricle are the result of exposure to extreme heat; stokers, smelters, braziers, puddlers and railway firemen are liable to circumscribed inflammations of the dermis on the anterior, the more

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exposed surface of the auricle and especially in the region of the helix and the tragus, while the dust and grime accompanying these and similar occupations, including metal, glass, stone and wood workers, using denatured alcohol, and grinders and polishers, mother-of-pearl cutters, brush makers, are frequently productive of some form of eczema of the auricle and of the external canal.

In all of the trades dealing with molten metals wounds and burns of the auricle are frequently possible. These injuries are usually circumscribed and, unless they penetrate to and involve a serious solution of continuity of the cartilage, are, usually, superficial in their effects; where, however, this solution of continuity has taken place, the intrinsic muscles of the auricle exert, by their contraction against lessened resistance, a distorting effect and the resultant deformity of the auricle is enhanced.

In the major injuries of the auricle the three bundles of extrinsic muscles, attaching and supporting the auricle, the attollens, attrahens and retrahens aurem, have to be taken into consideration, for under conditions in which the auricle has been nearly severed from its ligamentous and minor muscular attachments together with division of its mainly supporting muscular bundles, the attollens and retrahens aurem, it is necessary in effecting surgically reapposition of the wounded parts to stitch the severed muscles as well as the surrounding soft tissues in order to insure the lumen of the reconstructed external auditory canal, failure to resupply this muscular support of the auricle resulting in faulty apposition of the disrupted cartilaginous portion of the external auditory canal.

Blows upon the auricle incident to the trades and to some sports, boxing and football for example, are frequently productive of othæmatoma, a subdermal or subperichondrial sero-sanguinolent effusion occurring upon the anterior surface of the auricle to the obliteration of the superior fossæ. Similar effusions of lesser extent and occurring usually in the concha and lower portion of the auricle are the result, sometimes, not of directly applied violence but of strain in the lifting of heavy weights or of violent exertion as in running, the occurrence of the effusion in such instances usually implying some structural weakness in the cartilage or in the blood supply of the auricle.

In all forms of othæmatoma the thorough evacuation of the sac cavity and the permanent apposition of its walls are necessary to avoidance of the disfiguring distortion of the auricle evidenced not infrequently to-day as well as portrayed in the busts of ancient gladiators.

Injuries not only to the auricle but also to the external canal and even the drum head are not infrequent in the trades which have to deal with the fabrication of metals; blacksmiths, and other hammerers and forge workers are subject to injury from flying particles as are also stone masons and, less frequently, wood workers, while the dust resultant in these and similar occupations settling upon the auricle and about the entrance of the external auditory canal and requiring vigorous washing for its removal may be, in the course of that process, washed into the external canal and there retained

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with the effect not only of obtunding the hearing but of setting up irritation in the skin lining the canal. In this latter respect the character and constitution of the dust has a determinative effect, the dust of iron, steel and of coal having usually a merely obstructive effect, while that of zinc ore, containing also lead, cadmium and arsenic, is more or less irritating and consequently productive of eczema of the auricle and canal and of furunculosis and diffuse dermatitis. In wood polishers and jig sawyers where denatured alcohol has been used in the dressing and finishing of the wood or where acids have been used for checking and staining, the resultant dust is more irritating than that of the raw wood alone.

Wounds of the external canal from the entrance of foreign bodies occur, in the majority of instances, upon the posterior-superior wall of the canal at the junction of the cartilaginous and osseous canal, this region being particularly exposed to the impact of stiff foreign bodies thrust into the canal in the line of its long axis and from in front, such injuries having been recorded in game beaters and hunters from the entrance of twigs and in reapers from grain straws.

The stoppage of the external canal by accumulation of dust is materially guarded against by the growth of fine hairs projecting across the lumen of the canal; smeared as they are by the sticky and tenacious product of the cerumenous glands they afford an efficient protection unless, by forcible washing and pressure inward, into the canal, the accumulated dust, mingled with the natural secretion is thrust beyond them or they are artificially removed in ignorance of their protective value. The custom of barbers to cut hairs growing from the inner surface of the tragus and about the entrance of the external canal not only removes a valuable protection but allows the canal to become filled with the cut ends of the hairs. The frequency of inflammation of the external canal in the Chinese and some other oriental peoples is due to the mischievous manipulation of the public barbers who use a slender razor to shave the hairs in the external canal, wooden and metal scoops or curets to remove particles of dry skin, and pellets of ducks' down to wipe out the canal. All of these instruments, being unsterilized and indiscriminately used, not only mechanically irritate and inflame the delicate dermis but infect it, sometimes with serious results.

Mycosis of the external canal, usually aspergillus nigricans or flavescens, is found in weavers, spinners, wool sorters and other dust workers handling oily substances as well as in persons working in a moist atmosphere, paper mill tenders and laundry women for example, the condition favorable to the development of the spores of these and similar fungi being an oily or fatty soil and a warm moist atmosphere. The instances of this disease as described by Wreden in 1868 occurred in Russian cavalrymen who anointed their hair with grease and slept in the moist foul air of the barrack stables with their horses. The aspergillus is exceedingly tenacious in the hold of its mycelium upon the skin, causes much irritation and is disposed of only by sedulous

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cleansing and drying of the implicated skin area; repeated attacks of myringomycosis aspergillina sometimes cause a thickening of the dermoid coat of the membrana tympani and consequent impairment of hearing.

The disorders of the membrana tympani incident to the trades are usually of traumatic origin while the disturbances of function of the remaining portions of the sound transmitting apparatus may also be the result of sources of irritation in contiguous and communicating parts, most commonly the mucosa of the nose and nasopharynx.

Aside from direct application of force to a limited area of its surface, as in punctured or incised wounds of the membrana tympani, the more serious injuries of this membrane incident to the trades are those which come from sudden and considerable variations in air pressure and from disruptive force applied to the periphery of the membrane through the medium of the bones of the head. Sudden atmospheric condensation incident to unexpected explosions in mining, blasting and tunnelling, and the proximate discharge of artillery, especially in enclosed spaces, may cause so sudden an excursion of the membrana vibrans as to result in its rupture. This rupture thus produced usually occurs along the line of the long process of the malleus, the line of greatest excursion and most immediate check; ruptures of this sort are usually irregular in outline, the torn edges bleed freely and secure, by coagulation of the blood, an apposition of the edges with resultant speedy healing which is less likely to be the case when the wound is the product of direct contact and possible infection. The greater frequency of concussive ruptures of the drum-head in the navy, as compared with land artillery service, is due to the condensation of the concussive effect in the circumscribed spaces on shipboard, in the turrets and shields; the same is true of the concussions occurring in mining operations as compared with explosive occurrences in open trenches or on a level in the open air.

A similar rupture of the membrana tympani may be induced by a blow upon the head, especially if this occurs on the parieto-occipital angle of the opposite side the effect, by contrecoup, being to disrupt the inelastic structure at the side of the circle opposite to that at which the force is applied; except in so far as the middle ear is implicated, or an inflammatory process follows, the simple rupture of the drum-head is liable to spontaneous recovery, but if the apposition of the edges is incomplete, healing may be enhanced by the application of a thin paper dressing, applied on a moist cotton-tipped probe and allowed to remain in place, its removal being ensured by the normal progressive growth of the dermoid coat of the drum-head and of the external canal.

Another example of the effect of variation in atmospheric pressure is to be found in caisson workers, divers and aviators; in the former the continued exposure to increased atmospheric pressure with the subsequent return to normal pressure, if incautiously and too rapidly made, results in circulatory changes in the middle ear, impairing the hearing often quite as much as a

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solution of continuity of the drum-head incident to the still more rapid change in air pressure constituting a concussion.

The ruptures of the drum-head incident to diving, in the open air, are commonly the result of accident produced by sudden condensation of air in the external canal at the moment of striking upon, or entering, the water while congestion of the tympani mucosa and limited areas of effusion into the tissues of the drum-head with the creation of serous or hemorrhagic bullæ upon its outer surface are the manifestations more frequent in bell divers and submarine workers in armor.

In aviators the rapid alterations of altitude, productive of circulatory changes in the middle ear and labyrinth, as evidenced by the variable subjective noises, the resultant nervous strain with the accompanying secondary contraction of the tensor tympani muscle and the subjection to the continuous noise of the motor are all conditions which prove prejudicial, if only temporarily, to the hearing power, and when frequently repeated entail permanent results.

Variations of atmospheric pressure, moreover, in any of these occupations, if the change is rapid, are liable to excite reflex symptoms of nausea and vomiting through the medium of the effect more immediately produced in the irritation of the end apparatus in the semicircular canals. Protracted exposure to loud sounds of either very low or very high pitch is liable to excite the same reflex symptoms, tones of low pitch being more readily conveyed through the medium of the earth and of the body than the tones of high pitch which reach the sensorium more readily through the medium of the air and the sound-transmitting mechanism of the middle ear.

In all of the trades which entail more or less continuous exposure to heated air laden with dust and foul with poisonous gases the effect upon the ear is secondary to that induced in the mucous membrane of the nose and nasopharynx and varies, according to the degree and character of the irritation or infection, from a non-suppurative progressive thickening process in the soft tissues of the middle ear to a suppurative process more or less destructive in its course.

The number of the trades in which these two forms of implication of the middle ear are observable is large and their enumeration includes almost all of the occupations which are conducted in a dusty atmosphere and those especially in which dust and irritating vapors are among the by-products of manufacture; when to these unfavorable conditions there is added the loud noise of continuously operative machinery the cycle of injurious effects, so far as the auditory transmitting and perceptive apparatus is concerned, is complete and the welfare work of the trades will not have become adequate to its purpose until it has eliminated both dust and noise as essential factors.

In addition to the obtunding effect, in the external auditory canal, of dust accumulations, either spontaneously deposited or impacted by washing,

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with varying irritative results, according to the character of the dust, distinction in degree may be made in considering the consequences, to the middle ear, of continued occupational exposure, of the mucosa of the nose and nasopharynx, to dust of varying qualities and to atmospheric conditions varying in regard to temperature and moisture of the air.

In mining operations, for example, especially in the lower levels, the temperature is that of the tropics and the miner pursues his strenuous work under conditions which subject the mucous membrane of the upper air passages to a moist air laden with dust particles more or less irritating according to the character of the mining operation. The moisture in the air of the level and chambers in coal mines tends to throw the dust down or to minimize its irritative effect upon the mucous membrane, the disturbances of the upper air passages in miners being less in proportion than in the cases of miners or above ground workmen in dry dust laden air.

Roepke, who made personal observations, describes a coal miner as coming to the surface at the end of a day's work, with the nose and the nasopharynx coated with moist coal dust but, in a few hours, entirely free from obstruction and the mucous membrane relieved of its layer of coal dust, normally secreting and free from excoriation.

The percentage of implications of the mucous membrane of the upper air passages and the middle ear in other forms of mining in comparatively dry air varies with the character of the ore, the drier air and the more penetrating dust particles being productive of a more irritating effect than that which accompanies the mining of both the soft and hard coals; rock dust, more particularly that containing metallic particles, is especially irritating, the most pronounced cases of disturbance being found in the miners excavating the silicates of copper, of zinc, siliceous quartz, slate rock and the arsenical ores. In cases of exposure to arsenical ore dust the implication of the mucous membrane, especially that of the nose and nasopharynx, seems to bear a relationship both in frequency and in severity, to the proportion of arsenic in the product mined, suppurative nasopharyngeal catarrh and, secondarily, suppurative middle-ear inflammation being not unusual, the nasal mucous membrane covering the cartilaginous septum showing a predilection as a locality for the resultant circumscribed swellings and ulcerations. In the more chronic cases of arsenical poisoning Lewin has reported inflammation of the internal ear as a sequence of the evidence of arsenical poisoning in the nasopharynx and middle ear. In mercury mining the initial lesions occur most commonly in the mouth, the nasopharynx being comparatively unaffected but in the more severe cases of mercurial poisoning Wolf has observed acute labyrinthitis accompanied by Ménière's complex of symptoms.

In all mining operations there must always be taken into consideration, in addition to the air contaminations of natural origin, those which are artificially produced as a part of the process of mining, adding the fumes resultant from the explosion of gunpowder and dynamite and also endanger-

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ing the ear through the concussive effect of the explosion confined within a limited space.

In all of the chemical industries the similar contamination of the air by the liberation of acid fumes becomes a factor in the production of nasopharyngeal irritation and consequent implication of the middle ear while the various dust-producing trades, exercised above ground but in the circumscribed spaces of factories, mill rooms, and workshops, vary in the degree of irritation produced according to the constitution of the dust and its ponderability. The coarser dusts resulting from jig sawing and other forms of wood working would be less deleterious but for the acids and oils and varnishes with which the wood has been permeated or covered in previous steps of the process toward the finished product, while the weight of the finer dust resulting from metal, stone, and glass cutting, grinding, and polishing, incisive and irritating in itself, makes it less prejudicial to the artizan because of lesser prolongation of suspension in the air.

The exceptions to this rule are the trades in which the worker is in close relationship to a machine producing a dust of a mechanically irritating quality and of little ponderability, such as metal burnishing, glass grinding and mother-of-pearl cutting, lime burning, fertilizer mixing and packing and also the trades dealing with wool, hair, and grass sorting, packing and upholstering, flax heckeling, jute and hemp spinning, in which the infectious nature of the dust favors the inception of suppurative disease.

Under modern processes of manufacture the majority of the trades are conducted through the intervention of machinery interposed between the workman and the material employed with the resultant introduction of another prejudicial factor, especially and continuously invading the organ of hearing and even, in some instances, affecting that other portion of the internal ear which is a peripheral organ of equilibration, it being clinically observable that high grades of impairment of hearing are more common among those metal workers whose day labor confines them to subjection to intense sounds made up largely of overtones of high pitch than among the workers in noises of lower pitch, beamers in cotton mills for example.

Gottstein and Kayser found marked impairment of hearing in 50 per cent. of smiths and machinists examined by them. Holt found marked impairment of hearing in 35 per cent. of coppersmiths, and Barr found normal hearing in a little over 9 per cent. only of boiler makers, while Habermann reports that out of 31 boiler makers there was not one with normal hearing.

The consensus of opinion is to the effect that the degree of impairment of hearing in all these cases had a definite relationship to the duration and the intensive character of the operative sound. Both ears were usually tested, but more particularly that which was habitually directed toward the sound source. Subjective noises were an almost unvarying accompaniment of the impairment of hearing but dizziness or vertigo was present only in the more advanced cases, where the upper tone limit in hearing was notably decreased.

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Dizziness is also more likely to be an accompanying symptom, in mill and factory operatives, when to the noise of the machinery there is added a sensible vibration.

The majority of authorities dealing with this subject regard the affection of the hearing incident to exposure to loud sounds of high pitch as one of the internal ear solely, but Holt expresses the opinion that changes in the middle ear and sound transmitting apparatus are mainly the cause of the inpairment of hearing and Gradenigo makes three differentiations: labyrinthitis without middle-ear implication, chronic catarrhal middle-ear disease with labyrinthitis, and chronic advanced middle-ear inflammation with implication of the labyrinth and vertigo as an accompanying symptom; while Röpke is of the opinion that the majority of the cases of impaired hearing consequent upon exposure to loud noise are a combination of a disturbance of the labyrinth and of a catarrhal process in the middle ear, and Urbantschitsch reports a case in which the concussion of the labyrinth was accompanied by contraction of the intrinsic muscles of the middle ear.

These occupational sounds are divisible into two classes: sounds of sudden access and considerable amplitude, detonations, for example; and sounds of prolonged duration, intensive, of high pitch and replete with metallic overtones.

In all the clinical observations in regard to the effect of sound waves of considerable amplitude aerially conveyed, as a part of the occupational inroad upon the organ of hearing, the evidence is mainly in favor of the supposition that the principal channel through which the internal ear is subjected to injury from this source is that through the medium of the sound-transmitting apparatus of the middle ear and not through other bodily channels; this view being further supported by the fact that it is not the tones of medium low pitch, those most readily transmitted by bone conduction, which are the operative factors in the causation of the progressive impairment of hearing in the majority of the cases of occupational origin, but those of comparatively high pitch which find their access to the internal ear through the sound-transmitting apparatus only because of the obstacle presented to the passage of their short sound waves through the soft tissues of the body in which their impulse is almost immediately lost.

The mass of reported clinical observations upon this subject has found its more intimate support in physiological experimentation only within a few years and that by a series of investigations made by individual observers, but often in collaborative relationship, their purpose being the exact determination of the effects produced in the acoustic labyrinth as the result of subjection of animals to the continued influx of loud noises of different kinds.

The majority of these experiments included the use of very high pitched tones produced by whistles, organ pipes, high-pitched metallic bodies of various kinds, and metallic sounds of lower pitch under resonance conditions simulating those of the trades as exhibited in factory operatives, machinists,

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and boiler makers, the general results of these observations being that the exposure of one or both ears continuously, for a period of several days or weeks, to a pure tone of high pitch or to a mixed tone of medium low pitch, with correlated high-pitched overtones, was followed by a degenerative process in the organ of Corti, beginning in the nucleated ciliated cells, progressing to the neuron, and then, secondarily, attacking the vibrating mechanism and extending to the contiguous membranous labyrinth.

The degenerative process was found to bear a measure of relationship to the intensity and duration of application of the invasive tone and to be situated in corresponding relationship in the upper part of the first and the beginning of the second whorl of the cochlea.

Where one ear was closed to the extent of preventing the entrance of sound waves through the normal external passage, the cochlea exposed to the sound waves was alone found to have undergone a degenerative process. Von Eichen, for example, stopped the left ear air-tight in one experiment and, subjecting the animal to a whistle tone of C5, found only the right labyrinth affected by the characteristic degenerative process, the left labyrinth remaining normal. Care was taken, in the majority of the experiments, to transmit these experiment tones aerially only and not through the medium of the cage or box in which the animal was confined and consequently through the body.

Gruenberg exposed the right ear of a pigeon from 7 to 14 days to the continuous tones of a Bezold-Edelmann whistle A3, A4 with resultant evidences of degenerative processes in the papilla acoustica up to one-quarter the length of the ductus cochlearis, the left ear remaining normal.

Hoessli used, as the container for his animals, a suspended section of an iron water pipe, struck by automatic hammers to reproduce the conditions to which boiler makers are subjected, one ear being closed by cotton and collodion and, in some instances, both ears, in order to determine the effect of the sound corporeally conveyed. Where one ear was closed the degenerative process was exhibited in the ear remaining open; where both ears were closed the degenerative process was not exhibited, the observer concluding that air conduction was the only medium through which the acoustic effect was produced and that the ossicular chain is a conductive and not a protective mechanism.

In concurrence with the determinations of other observers Hoessli found that the labyrinth lesion occurred in relatively the same area in the cochlea in all the animals subjected to the influence of a continuous tone of high pitch, and that the higher the tone the lower was the area of lesion in the cochlear tube, while Habermann, in the observations made upon boiler makers and blacksmiths, found that the degenerative alterations in the organ of Corti, in the nerve, and in the related ganglion cells was more extensive in the basal whorl, the upper portion of the cochlea remaining normal.

Wittmarck found that with intense sounds of high pitch and even of short

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duration the corresponding area in the cochlea was affected, in contradistinction to other observers, but placed especial stress upon the influence exerted by influx of sound corporeally conveyed.

Marx is of the opinion, from a corresponding series of investigations, that the local degenerative process in the organ of Corti is, in proportion to the intensity of the high pitch employed, not only more pronounced but also more extensive in area; the high tones of a continuously blown whistle, A3 and A4, produced the recognized degenerative effects in the organ of Corti; corresponding tones of lower pitch A2 and E2, being less intense, did not result in degeneration.

In the detonation experiments of Hoessli only the upper portion of the first whorl and the lower portion of the second whorl were affected, a result consonant with the results of Siebenmann and others and with the clinical observations of Friedrich and Jaehne, while Yoshii reports, as the result of detonation with middle-ear injury, rupture of the membrane of the round window in but one case, the membranes of the oval and round windows remaining intact notwithstanding the presence of petechiæ and hemorrhage in the vicinity of the windows, there being corresponding changes in all parts of the organ of Corti, the outer and inner ciliated cells being swollen, distorted and sometimes separated from their base, the rods distorted and the intermediate space filled with cellular detritus.

Clinical experience has shown that with continued exposure of the human subject to intense sounds of high pitch there is evoked, in addition to a sense of fullness, subjective noise and malaise, a greater or less degree of dizziness; and the laboratory evidences of the location of degenerative changes in the lower portion of the cochlea suggest the possibility, in view of the vestibular contiguity, of a possible secondary excitation of the equilibrating end organ.

While the demonstration of labyrinthine changes has been sufficiently precise and sufficiently extensive to emphasize the importance of prophylactic measures in reference to the noises of the trades, there is another question still a matter of debate and open to clinical observation for its determination, and that is the possible secondary implication of the sound-transmitting apparatus of the middle ear, a change progressively decreasing the conductivity in the channel through which the causative factor in the cochlear degeneration finds access to the internal ear.

Hensen, Bockendahl, and Pollak have reported observations of apparent reflex contraction of the tensor tympani muscle in response to high tones, and it is a not uncommon clinical experience to find in boiler makers, machinists, locomotive engineers and firemen, in addition to a decrease in hearing for tones of medium and high pitch, both by bone conduction and aerially conveyed, objective changes in the sound-transmitting apparatus of the middle ear evidencing a thickening of the tympanic mucosa and a contraction of the tensor tympani muscle, conditions referable in many instances to such other occupational influences as sudden changes in temperature and exposure to

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dust, and other causes inducing irritation of the nasal and nasopharyngeal mucous membrane.

In the earlier stages of impairment of hearing as the result of exposure to loud, continuous noise, clinical observation shows a depression of the membrana tympani consequent upon retraction of the tensor tympani muscle, this objective evidence appearing in advance of the thickening process in the middle ear.

Further observations in the cases of workers in loud noises serve to substantiate the idea that the muscular contracture and consequent fixation of the sound-transmitting apparatus, supplemented by the thickening of the tympanic mucosa, serve as an obstacle to the inroad of those loud and high-pitched sounds which have been shown, by animal experimentation, to find their access to the labyrinth, with deleterious effect, especially through the natural channel of audition.

Similar changes, more especially the muscular contracture, have been found in telephone operators, and are more probably the result of the sudden and unexpected movements of the telephone armature in response to current interruptions than of the normal, ordinary use of the instrument in listening to the human voice, measurements of the intensity of the voice at the receiving instrument, as compared with that at the transmitting end of the line, having shown so considerable a loss as to make the volume of voice received a very inconsiderable percentage of that immediately transmitted, while the concentration of the voice directly upon the ear of the operator, as is the case in the majority of the forms of apparatus including close adjustment of the telephone receiver, makes the determination of even a very moderate tone a matter of custom without effort except in so far as accommodation to the qualitative overtones of the consonant sounds of nearest resemblance is concerned.

Repeated observations, made at intervals, in the cases of telephone operators with normal or approximately normal hearing have shown that very little change in the hearing, even in years of service, results from the occupation, but that with changes in the sound-transmitting apparatus with appreciable diminution of hearing upon entering the service, a further diminution is noticeable after prolonged occupation in telephone work.

The demand upon the normal hearing power in telephone operation would seem, therefore, to be so little in excess of the usual conversational demand as to cause no resultant defect.

The sum of the recorded statistics of observations in this rapidly increasing branch of public utility service is too small to make other than very broad and very general conclusions possible, and the subject is one which might advisedly receive more attention on the part of the medical profession.

Among the most complete are the conclusions arrived at by Blegvad as the result of a series of precisely conducted observations, embodied in an extended record beyond the capacity of a brief review but well repaying

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careful reading, to the following effect: In 26.4 per cent. of the 371 telephone operators with normal hearing, who were made the subject of examination, there was a retraction of the drum-head in the ear to which the receiving instrument was usually applied, the other ear evidencing either none or only a slight retraction. The continued use of the telephone caused no depreciation of the hearing in operators with normal ears, nor was there, on the other hand, any appreciable increase in the capacity for the hearing of tones of high pitch and slight intensity, as is sometimes claimed by the operators, this claim being supported rather by the increase in the accommodative power for tones of this class and by the gradually acquired habit of eliminating mentally the coincident and the extraneous noises. The aural lesions and traumatic neurosis incident to the accidental introduction upon the line of strong currents are the result of the exhibition of a loud and sudden sound and are to be guarded against by proper protective construction in the telephone lines.

It is evident, therefore, that in addition to the occupational conditions which should come under the consideration of welfare work as they have been previously regarded, there should be especial attention given systematically, both by study of cause and effect and by effort at protection from the results of continued exposure to loud and especially penetrating noises.

The sound-transmitting apparatus of the middle ear serves the double purpose of sound transmission and of protection to the transmitting and perceptive mechanism beyond it, but is capable of exerting this office only for periods of limited duration and, under conditions of continuous subjection to loud sounds, covering a limited portion of the audible scale, in itself becomes fatigued and incapable of exercising its protective office. The obstacle presented to the passage of sounds of short wave lengths through the soft tissues of the body affords a certain measure of protection in one direction, and the obtunding of the external auditory canal prevents the influx of the objectionable sound through that natural passage, but this serves as only a partial protection and an important step in the welfare work of the trades will be in the elimination of that offensive and injurious by-product of mechanical action, sustained and unnecessary noise.

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DIVISION V

Occupational Affections of the Skin.

Cancer and Occupation. X-ray. Radium

CHAPTER I

OCCUPATIONAL AFFECTIONS OF THE SKIN

BY JOHN T. BOWEN, M. D., Boston, Mass.

Dermatitis Venenata.—The term dermatitis venenata has been given to that class of inflammations of the skin that is produced by external contact with irritating agencies, whether of a solid, liquid or gaseous nature. These agencies may be of very different character, and may belong to the animal, vegetable or mineral world. No pathognomonic symptoms or appearances are associated with this class of inflammations, although certain more or less general characteristics may, if present, serve to distinguish them. James C. White, in his classical monograph on Dermatitis Venenata, enumerates among these characteristics "mainly a sudden onset, a rapid evolution of primary lesions, some peculiarities in their situation within the cutaneous tissues, an unusual color in their fluid contents, a greater intensity of inflammatory action, and continuity of development within given areas, the localities attacked and the sharply defined limitations of the regions affected, a marked asymmetry and an artificial-appearing configuration in the eruption, *their occurrence in those employed in certain arts and professions*, and other unusual extraordinary appearances at times, which cannot be defined." On the whole the greatest number of cases of dermatitis venenata is produced by the action of plants possessing irritating properties, and foremost among these poisonous species may be mentioned the different varieties of rhus: poison ivy, poison sumach, etc. Other frequent causes of dermatitis venenata are the use of irritating cosmetics, and clothing which has been colored by poisonous dyes. The French writers often group these cases of dermatitis venenata under the broad conception of "éruptions artificielles," which includes feigned eruptions, eruptions of digestive origin, eruptions from external and internal medicaments, therapeutic eruptions, and professional eruptions.

Much emphasis should be laid on dermatitis venenata in a chapter dealing with the professional dermatoses, because by far the larger number of cases of these affections belongs to the former group. The so-called trade eczemas, affecting chiefly the hands and the parts exposed to contact with the irritating substances handled in various trades, are forms of dermatitis venenata, at least at the outset and etiologically, before, in other words, the element of

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chronicity and stubbornness that is associated with the term eczema has asserted itself. These so-called trade eczemas are the most common examples of occupational dermatoses and will be considered in this chapter under the eruptions peculiar to the various occupations.

Trade Stigmata.—Before describing the various occupational dermatoses that may properly be included under this title, a word may be said with regard to the so-called trade stigmata. This name has been given to those changes in the skin, caused by various occupations, which are not of sufficient pathological importance to be classed as dermatoses, and which may yet be quite typical and characteristic of the work in which the subject is engaged. They often consist in a simple coloring or circumscribed thickening of the skin due either to exposure to the sun or to the mechanical effects of constant pressure. Such appearances of the skin, while of no great consequence as regards their symptoms to the one by whom they are acquired, may be important from a medico-legal point of view. In the case of dyers and of those who work in aniline manufacturies, an obstinate staining may resist all efforts to clean the hands at the end of the working day. The strong alkalies and other substances that are often employed to aid in cleansing may be very injurious to the skin as will be later emphasized, and give rise to an active inflammation. Exposure to the sun's rays in the case of cabmen, sailors, soldiers, etc., causes the reddening and later bronzing that are associated with an out-of-door life, and that may often reveal the occupation of the subject by their intensity and localization. In this case the reddening and pigmentation are to be regarded as protective phenomena against greatly increased action of the sun's rays.

Thickening of the skin may occur either diffusely or in circumscribed patches, when constant pressure or friction is brought to bear on certain places in the performance of different kinds of work. The inner surface of the hands of those who engage in almost any kind of rough manual labor tends to become thickened, this being due chiefly to the piling up of the horny cells of the outer epidermic layer. From circumscribed thickenings, callous places, a hint may be given as to the occupation of the subject, and in this way the skin changes may be of value in medico-legal matters. Thus shoemakers exhibit callous patches on the flexor surface of the right hand from the pressure from the hammer, also circumscribed thickening in the sacral and coccygeal regions on account of their constant sitting. Tailors, blacksmiths and carpenters are affected with similar cutaneous thickenings at the site of pressure from the tools that they use, on various parts of their fingers. Analogous appearances are observed sometimes in musicians, those who play on stringed instruments showing callous thickenings at the end of the thumb and forefinger. A circumscribed thickening of the skin of the neck is occasionally observed in violin players from the constant irritation caused by the violin pressing upon this spot.

With regard to the discolorations of the skin, apart from the bronzing

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and pigmentations that occur in those whose occupation exposes them to the weather and to the sun's rays, there are several conditions produced by the penetration of foreign bodies into the skin. Blaschko has described an affection common in workers in silver, in which small spots from the size of a pin's head to that of a pea, of a bluish-black color, appear on the hands and fingers, especially on the dorsal surface of the left hand. These spots do not tend to disappear with time but are permanent, remaining of just the same appearance after they have attained a certain size. The bluish-black color is produced by the penetration of very small particles of silver through the epidermis into the corium, in the process of filing, sawing and planing. The silver is then dissolved in the alkaline tissue fluids and again precipitated in the form of very fine particles, which are deposited in the connective tissue and elastic fibers. Lewin regards the lymph channels as the place of deposition of the silver.

Similarly Elliot, in 1891, described a "pseudo-pigmentation on the hands of millers." His case concerned a man whose occupation was to smooth off with a steel chisel the roughnesses that occurred every day in the grinding stone, and on the backs of whose hands and fingers there were numerous dark discolored spots varying in tint from brown to black, some of them as large as a pea. These were caused by the small particles of metal that were chipped off constantly in the course of this work. The discolored spots were evidently caused by the oxidation of the small bits of metal, and it was shown that when the cause was removed, there was gradual elimination of the rust particles. These discolorations in millers have been confirmed by others, and the dorsal aspect of the left hand has been shown to be their most common seat. They have been used by Tardieu as a means of identification.

Occupational Dermatoses Proper.—In almost all the cases in which poisonous material is introduced into the economy through the skin, there is a fracture or opening of the integument, however small, at the point of contact. Tuberculosis, syphilis, glanders and other serious systemic infections are thus introduced, and there is no good reason for assuming that these noxious bacterial agents can pass through a wholly intact epidermis. An exception to this rule is found notably in the case of mercury, which, as is well known, may produce severe constitutional and poisonous effects after its absorption through the unbroken skin, and also in such cases may produce a universal and even dangerous form of dermatitis. Somewhat analogous to this dermatitis from mercurial intoxication is a form described as occurring in workers in aniline factories and in dye houses, which runs an exceedingly acute and virulent course. It starts as an acute dermatitis, beginning on the exposed portions—hands and face—and showing its most active characteristics in those places. From here it spreads to the arms, trunk and lower extremities. The affection may be confined to the erythematous stage, causing much œdema of the hands, ears, penis and scrotum, or

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it may progress to a quite intense bullous dermatitis, with large and small, often confluent, bullæ, followed by a long-lasting and profuse desquamation. When the feet are more acutely affected than the hands, it can usually be shown that the subjects have been working without stockings or other protective covering. This affection has a definite course and is followed by a marked idiosyncrasy to this form of poisoning. It is assumed that in this case certain toxic substances find their way through the intact epidermis to the deeper parts of the skin, expending their action especially on the vessels of the skin. Blaschko gives a list of 19 colors with which such a type of dermatitis has been observed, but points out that they must not be looked upon as generally poisonous substances, inasmuch as a certain idiosyncrasy seems to be necessary in the persons affected. Such forms of dermatitis have also been observed in the case of workers in quinine factories, in those who have to handle various foreign woods, satinwood, teak, etc.

Dermatitis, Usually of an Eczematous Type.—By far the larger number of cases of occupational dermatoses are exhibited in the form of a dermatitis of an eczematous type, and most of them come under the commonly used heading "trade eczema." It does not come within the scope of this article to discuss at any length the much mooted question of eczema, including the application and limitation of this term. Many of the cutaneous disturbances caused by the occupations produce an acute dermatitis very similar to, if not indistinguishable from, an ordinary dermatitis venenata, caused in the same way by the direct contact with poisonous or irritating substances. Most of these inflammations affect the hands, head, or whatever parts of the body may be exposed to direct contact with the irritating substance. Those who hold to the older view class these as cases of acute eczema, their recurrence and persistence, whether by continued exposure to the primary exciting cause, or without this objective incentive, placing them in the group of chronic eczemas. A very large number of the cases of occupational dermatitis begin, as will be seen in the course of this article, in quite a different way, going through no preliminary acute stage, but presenting the various clinical features common to ordinary cases of eczema to which no definite external causation can be assigned.

These cases of occupational dermatitis, or "trade eczemas," form an astonishingly large proportion of the total number of cases seen in the dermatological departments of hospitals. Fordyce has stated that at his clinic at the University and Bellevue Hospital Medical College about 2 per cent. of the total number of new cases for 1911 constituted occupational dermatoses. The great majority of these were of the type known as trade eczemas, and while a good many of them yielded readily to treatment, a considerable number proved refractory, and necessitated abandonment of the particular work for a time, or changing of occupation. Knowles reviewed 24,459 cases of diseases of the skin that had been seen by him during 9 years at the dermatological departments of the Pennsylvania University and Howard Hospitals.

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Of this number 4142 were diagnosticated as eczema, and of these 768 were from distinctly occupational causes. He points out that this estimate included only those cases where there was direct exposure from the occupation, and cases of eczema rubrum, for example, that were evidently produced by long standing at various occupations, were not considered.

It is a matter of common knowledge that not all persons who are exposed to cutaneous irritants are affected adversely by them. This is well seen in the exemption of many people from poisoning by ivy and other plants which may produce a severe dermatitis in others. In the same way not all the workmen exposed to the same conditions in their particular trade are similarly affected by them. Others, after once exhibiting a susceptibility, become increasingly sensitive to such irritating influences, as time goes on. As a rule, the occupational dermatoses do not occur suddenly, or at the outset of exposure, but after a considerable time, sometimes several years. This acquired sensitiveness may be explained either by the cumulative action of the constant irritant, or on the theory of a gradually diminished power of resistance.

It is an undoubted fact that the sensitiveness of the skin toward irritants varies with the individual. Also it is apparent that this individual sensitiveness is more pronounced in the case of exposure to some kinds of irritants than to others. There is comparatively little difference in the sensitiveness of various people to the action of thermic irritants. How to account for this varying susceptibility of the skin to reaction from poisons and irritants is by no means agreed upon. Thibierge and Jacquet have considered that general or bodily conditions have the same effect in producing skin inflammations as local conditions, and especially in respect to digestive disturbances. Of 27 cases of trade eczema studied by Jacquet and Jourdanet, in 22 there were errors in digestion; the subjects being either alcoholics, addicted to excess in tea and coffee, or accustomed to insufficient mastication and to hurried eating. In 25 of these cases, when these causes were removed there was improvement. Darier, also, considers that alimentary hygiene has a marked influence on the vulnerability of the skin. Four patients were cured or improved in 7 days by the treatment of the digestion without local treatment, and nine were cured or improved without suppression of professional contact. On the other hand, Herxheimer states that in a great many trade eczemas, and other chronic eczemas, observed in the course of years, no progress toward amelioration was made by attention to the diet alone. He does not consider it necessary to invoke trouble with the internal organs in trade eczemas, as there are many local factors, such as the varying fat content of the skin, the composition of the sweat, etc., that may account for varying susceptibility. On the whole, there is insufficient data from which to determine the underlying reason for this varying susceptibility of the individual. There is no foundation for the assumption that it implies a difference in the condition of the internal organs, or of the vital processes in general.

The number of different occupations that have been found to be the cause

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of cutaneous disturbances is very large. Herxheimer has compiled 74 different "trade eczemas" and asserts that probably some were overlooked, while new ones may be expected to arise constantly, with new methods of technique. Age and sex appear to be factors of very little importance in occupational dermatoses generally, although Merzbach's figures show that the ages from 20 to 40 are the ones in which subjects are most likely to be affected. In certain occupations, it is true, only women have been found to be affected, in others only men. Nationality is not a factor of importance. Knowles, from the investigations alluded to, found that a very large majority of the outbreaks occurred during the winter months, in practically all of the occupations concerned; also that most of the cases collated by him lasted over a period of weeks or months independently of the abandonment of the irritating occupation; that the eruption had a tendency to relapse, and that a less amount of irritation was needed to excite the later attacks.

OCCUPATIONAL DERMATOSES, CONSIDERED ACCORDING TO THE VARIOUS TRADES AND CALLINGS

Housemaids and Houseworkers.—People engaged in these occupations contribute a very large number of cases to the class we are discussing. The type of eruption usually presented is that of an eczema, most commonly of the squamous or vesicular variety, but not infrequently papular, erthematous or oozing in character. In almost all cases the dermatitis is present on the hands and arms, spreading oftentimes to the legs, feet, face and neck. Extensive eruptions may have their origin in this way. The direct cause of most of these cases is the constant use of water and soap. Water and soap are both cutaneous irritants, withdrawing the protecting fat and softening and removing the outer horny layer so that the skin is deprived of some of its power of resistance and becomes more vulnerable. Washerwomen, who are included in this class, are especially liable to a dermatitis of the hands and arms from the continual immersion of these parts in soap and water. The strong alkaline soaps that are used on account of their superior detergent powers are responsible in great measure for these irritating effects. Various washing powders and cleansing alkalies are added oftentimes, especially for the greater facility in washing clothes, and these are to the highest degree irritating. The poor character of many of the soaps is also responsible, a free alkali when present adding much to the irritating properties. These results from the use of soap and water, so common in houseworkers, are often seen in people who are not obliged to work, but have acquired the habit of constantly scrubbing their hands with soap and water. Fordyce states that nearly one-third of the occupational diseases seen in his clinic during a year were in people whose work caused them to employ soap and water freely, or else the various alkaline cleansing agents. Knowles found that in his experience housewives who had to do all of their own work on account of

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financial reasons, such as cleaning, washing and cooking, were the ones most likely to be affected in this way. Another affection to which people of this class are more or less subject, together with all those whose occupations require them to stand constantly, is chronic dermatitis of the lower legs, combined oftentimes with varicose veins and resulting not infrequently in the well-known stubborn varicose ulcer. In washerwomen, besides the inflammation that has been described, a peculiar condition of the face is often met with. This is characterized by the presence of a number of rounded, translucent yellowish-white vesicles, of about the size of a small pea on the average, which are called hydrocystomata, and which represent a cystic condition of the sweat ducts of the middle third of the face. These lesions also occur sometimes in those who perspire freely, and in the case of washerwomen are attributed to the warm moist atmosphere in which the subjects work.

Bakers.—A dermatitis of the hands and forearms of bakers, caused by constant contact of the skin with the moist dough, aided by the heat of their surroundings, has been frequently observed. Knowles remarks that "bakers' eczema" or "bakers' itch," as it has been called, was first referred to by Willan in 1817 under the name of psoriasis diffusa. Knowles reports that he has met with 11 cases of this affection. Although no accurate figures are available, it is no doubt true that a large number of cases have been observed in the skin department of the Massachusetts General Hospital during the last 20 years. The lesions present the appearance of rather circumscribed crusted patches, often circinate in shape and resembling psoriasis somewhat. They are most common upon the hands, and are often similar in character to the appearances seen in workers in sugar. Acne, furuncles, and localized abscesses are said to be of frequent occurrence among bakers, the last two named being doubtless from the abundant opportunity for secondary infection afforded by the situation and character of the dermatitis. It is quite unlikely that the presence of mites in the flour is to any degree responsible for the eruption.

Barbers.—Barbers and hairdressers are exposed to much the same external influences as are washerwomen and houseworkers, from the necessity of constant contact of their hands with soap and water, in the process of rubbing in the soap for shaving and in shampooing the head with various soap preparations. They often use alcoholic solutions of the potash soaps, employed on account of their greater detergent qualities. The various stimulating lotions that are rubbed into the scalp are also often the cause of irritation, such as strong cantharidal preparations, the various aniline hair dyes, quinine hair tonics, etc. The resulting dermatosis usually takes the form of a papular eczema confined to the hands, and especially affecting the fingers, although the arms also may be involved. Knowles has collected 15 cases of this affection. Fordyce has seen unusually severe forms of eczematous dermatitis in barbers who had been in the habit of using aniline hair dyes on their customers.

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Workers in Sugar, Chocolate and Candies—Confectioners.—A dermatitis of the hands and arms of those who were in the habit of handling sugar was one of the first forms of trade dermatoses recognized. White says, "The old-fashioned term 'grocer's itch' is rarely applicable in these days of refined and double-refined sugars. Formerly, when brown sugars were almost universally used in the household, it was employed to signify an eczema of frequent occurrence upon the hands of the grocer who handled not only these but many other substances belonging to his trade, which were more or less irritating to the skin. What share the sugar had in thus exciting the skin, it is impossible to say, but to it was attributed the greatest. These sugars often contained an abundance of mites, but it is improbable that they excited any irritation by contact."

Eruptions of an eczematous type on the hands of those who work in sugar and candy manufacturies are frequently observed, and are well known to all dermatologists who have had experience in public clinics. The eruption may at times extend to the arms. Knowles has noted an outbreak in four female candymakers and in two of the male sex, three working in a sugar refinery, and one a chocolate roaster. He considers it difficult to determine the exact causation in these cases, but the trade was undoubtedly responsible for them, as the condition relapsed whenever the subjects resumed their occupation. At one time numerous cases were seen by the writer in girls whose occupation was to dip candies in chocolate in order to produce a coating of the latter. These eruptions usually took the form of a papular dermatitis. As White says, another cause than that of the sugar is present here, viz., direct heat and general high temperature of the workshops. Rémy and Broca have reported four cases of ecthyma of the arms and legs in people who worked in sugar refineries, either naked or partially so. These eruptions they considered due to contact with the sugar, as they affected the uncovered portions of the body, beginning with a pustule, and often being associated with an eczema. These cases were found to be especially common in the workmen who had recently arrived, and to be very uncommon in those who practised frequent washing and paid strict attention to hygiene. In all cases it occurred in those whose skins had been in contact with melted sugar or molasses, or whose trousers had become impregnated with these fluids. In these cases of molasses workers, it is common to work in the saccharine fluid up to the waist. Fordyce refers to a personal communication from Winfield in which he speaks of a dermatitis in sugar refineries, occupying the hands, forearms and legs and suggesting scabies somewhat.

Poncet, Albertin, Chaussende, and other French writers have described a form of inflammation of the nails occurring in confectioners. This has been observed chiefly in the workmen employed in the great factories of southern France in making sugared fruits. The eruption has been attributed by many to the chemical acids contained in the juice of the fruits, into which the hands are dipped; others consider that it is due to the saccharine solutions

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and that it is analogous in its causation to the usual "sugar bakers' eczema." There can be no doubt that the cold and hot water into which the fingers are constantly dipped acts as a contributing factor, at least, in the etiology. The affection begins in the form of erosions and fissures about the nail fold, followed by inflammation, ulceration and granulations, together with a sero-purulent secretion. The nail is often loosened from its nail bed and is destroyed. The course is very chronic, lasting for years, with subacute intervals. The characteristics are: its affecting a number of the nails simultaneously, the discoloration of the nails, the granulations about the nail fold, and the flattening of the nail phalanx. Poncet considers that the onychia is sufficiently characteristic to warrant its serving in legal medicine, as a means of identification. The middle and ring fingers are those first affected. After the fall of the nail the extremities of the fingers take on a characteristic form, a spatulate shape, which is lasting. Strauss has reported three cases of this affection, together with a good description.

A lymphangitis has been described by Oliver as occurring in workers in sugar refineries, among the refiners and molasses stirrers. A slight degree of constitutional disturbance has been noted, and furuncles are sometimes an accompaniment. Gaillot has attributed the cause of this lymphangitis to the staphylococcus pyogenes aureus which is found in the residue of the molasses, acting in conjunction with the temperature of the factory and the condition of the skin.

Builders, Masons, Brickmakers, Laborers.—This class of people are subject to dermatoses from the character of their occupation, as well as from the exposure to the weather incident to their calling. Excessive cold, beside the various degrees of more or less generalized dermatitis that it may produce, is the cause in many instances of chilblains, which often produce much suffering and may interfere seriously with the occupation. In this connection the rosacea so common in drivers, coachmen, and those whose faces are much exposed to the action of the wind and weather, may be alluded to. It is also true that much of this may be attributed to the effect of alcohol, to which this class of people is notoriously addicted. Varicose veins with their sequel, chronic ulcer of the lower legs, is also a prominent affection in these callings, as well as in all others in which long hours of standing at work are necessary. Builders and masons are very often affected by a dermatitis of the exposed parts of the body caused by the irritation from the lime and cement with which they have to do. Plasterers, whitewashers, and paperhangers are similarly affected. Brickmakers are often affected with a dermatitis of the palms.

Tobacco Workers.—Long-continued, stubborn eruptions upon the hands are observed in those who work constantly with tobacco, in the process of cigar making as a rule. These eruptions are very liable to relapse. The dermatitis has by some been attributed to the irritating effects of the nicotine, by others to the caustic solutions that are used for separating the tobacco

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leaves. Tobacco workers generally work under very unfavorable hygienic conditions, and the heat and moisture to which they are subjected must be looked on as prominent factors in the etiology of this dermatitis.

Furniture Polishers.—This trade lends itself with a surprising frequency to a well-marked and rather characteristic dermatitis of the hands and especially of the fingers, the so-called "polishers' itch." It is attributed to methyl or impure alcohol which is used in making various varnishes and polishes, and impure benzine for cleaning purposes. A somewhat characteristic appearance is produced in these cases from the staining of the nails and skin by the preparations used, and by the odor that in recent cases clings to the epidermis, sodden and impregnated with the preparations. Not infrequently the writer has noticed a dermatitis of the face also in these cases, of an erythematous type, situated especially about the mouth, nose, and ears, and of a peculiarly shining, glistening aspect. Also in many cases there has seemed to be a condition of general anæmia, and lowered physical vigor. Machinists and oilers may also be similarly affected. Blaschko states that before the introduction of denatured spirit (*i.e.*, before 1879) there were very few skin affections in furniture polishers. He refers especially to troubles with the eyes, catarrh, and pulmonary affections in this class of subjects. After a careful search of the other substances used by furniture polishers, Blaschko concludes that the pyridin used in denaturing the alcohol is possibly concerned in the causation of the dermatitis.

Bartenders and Liquor Dealers.—Knowles reports eight cases of dermatitis of the fingers, hands, and forearms in bartenders, one in an alcohol dealer, and another in a brewer. The mechanism is much the same as in the case of housemaids and cooks, constant immersion of the hands in water while cleaning glasses and the strong soaps used, causing a dermatitis of the hands and forearms that is often very intractable, especially if the occupation is persisted in.

Printers and Workers in Metals and Minerals.—Numerous instances of dermatitis in workers at these trades have been reported, and are commonly met with in dermatological clinics. Knowles records eight cases in printers, two in typesetters, one in a press-room worker, one in a handler of the dye presses, one in an architect, one in a draftsman, and one in a newspaper seller. He says further: "The helper around a type foundry is apt to have an outbreak because of the irritating oils he is forced to handle. The electrotypers handle lye to wash off the forms because of the graphite and the dust. They take a mould, consisting of plumbago and wax, and place it in a wash of bluestone and muriatic acid, an electric current is then turned on, and a copper coating is formed on this wax 'shell.' This shell remains in the solution for from 40 to 45 minutes. This mould is then placed in a machine and lead is run in on it, the wax is then removed and the electrotype is finished. Stereotypers use a lead mould without the copper surface. The compositors handle only gasoline or benzine to clean the type. The eruption is therefore

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more apt to occur in the electrotypers, the stereotypers, the helpers in the foundry and in the makers of the moulds than in the compositors."

Zellner and Wolff report that there have been a great many cases of skin affections of very similar character occurring during the year 1912 among printers employed by Berlin newspapers. The first appearances were those of a subacute dermatitis of the hands and forearms, somewhat artificial in type. It was found upon enquiry that the workmen themselves attributed these affections to certain substitutes for the oil of turpentine, which had heretofore been used for the purpose of cleaning type. It is stated that of late benzine, lye, petroleum and certain kinds of pine oil have been used as cheap substitutes for turpentine oil in removing the ink from printers' forms. These substances are either directly poisonous to the skin themselves or contain irritating impurities. An inferior benzine, which it has been found is often used as a substitute for turpentine oil is decidedly irritating to the skin. This substance is also being used in certain cheap kinds of paint. Thirty-seven samples of these substances used for cleaning were analyzed, with the result that 32 or 87 per cent. of them were found to be harmful, and a return to the use of a high-grade oil of turpentine is therefore considered imperative from the point of view of the workmen's health. As illustrating the poisonous character that may be present in some grades of benzine, the testimony of Dr. Oestreicher of Berlin is adduced, who has seen eczema caused by wearing gloves that had been cleansed by benzine, and he further states that harmful skin effects have been caused by cleaning the skin with benzine before operations, and also by using this substance to remove surgical and dermatological plasters.

Workers in metals and minerals, especially those engaged in the silver and electrotyping trades are very subject to a dermatitis of the hands and forearms. Hall, quoted by Knowles, has described the mechanism by which workers in these trades acquire an eruption of the type of a papular or papulovesicular eczema. The goods are cleaned by means of a revolving brush, on which sour beer drops from a trough above, and the liquid from this revolving brush is splashed upon the hands, forearms and sometimes faces of those engaged in the work, acting thus as a more or less acute cutaneous irritant. The same writer describes an outbreak of dermatitis on the hands and arms of polishers and burnishers of silver, where the irritation is due to the "rouge" used for burnishing, this so-called "rouge" being a mixture of "quicksilver," iron and wax. There are various grades of this "rouge," the coarser variety showing greater powers of irritation. Bichromate of potash is said by Crocker to be used by French polishers and to be productive of a dermatitis frequently. The cyanide of potash is also used for cleaning silver and gold, and may produce cutaneous trouble. Knowles cites several cases in point.

Dyers and Aniline Workers, Dressmakers and Photographers.—For years severe cases of dermatitis have been noted in dyers and those employed in

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establishments for the manufacture of aniline colors. Compounds of arsenic, mercury and chromium have been used in making these brilliant dyes from coal tar—substances that are of themselves powerful cutaneous irritants, and are to be regarded as in some measure responsible for the dermatitis common in those engaged in this trade. Bichromate of potash when applied to the skin produces an eruption of papules and pustules, and later deep sloughs and ulcers. Hence those workmen who use this substance in their trade—dyers, electricians, photographers, etc.—often exhibit deep and painful ulcers of the hands and arms. Harrington has published the case of a capmaker who was poisoned on the face, neck and hands after cutting up a piece of dark blue cloth for boys' caps. Analysis showed the presence of a large amount of chromium. Two years afterward a similar eruption appeared after dusting and packing away the same unmade caps. Poisoning from the aniline dyes and bichromate of potash is by no means confined to those working in these substances. Numerous instances occur of varying grades of dermatitis in the wearers of stockings, gloves, caps, underwear and shoes which have been dyed in this manner. White reports several cases of deep-seated dermatitis produced by gloves and stockings of quiet colors that may have been due to the presence of chrome mordants. Other instances of analogous methods of poisoning are in those who cut paper flowers and patterns from paper impregnated with aniline dyes, upholsterers, milliners and dry-goods clerks, shirt-makers, etc. Aurantia or hexanitrophenylamine is a dye that has often been used for coloring cheap yellow leather shoes. Crocker states that those who work with it are liable to a severe dermatitis of the hands, as the liquid is sponged on to the leather to be dyed.

Among photographers numerous instances of dermatitis have been observed which were attributed to bichromate of potash, and Richardson of London has described these cases under the title of "the bichromate disease." Beers reports cases of dermatitis among photographers due to the irritating effects of metal. Knowles produced a series of four photographers and one photo-engraver afflicted in this way, and has also referred to the case of a moving-picture operator poisoned from handling the films, carbon, and cement.

Rident has noted cases among the linen-dyers in the cloth manufactories of Elbeuf who dip the linen in a bichromate solution. As a rule, after working from 5 to 6 weeks with the hands immersed in this solution, redness, vesiculation and oozing of the backs of the hands follow.

The dermatitis that occurs upon the hands and forearms of tanners may be alluded to in this place; this is due to the chemical process through which the hide and leather are passed. Knowles thinks that the bath of bichromate of potash and muriatic acid in which the hides are placed, and in which the workmen's hands remain for a considerable time, is the cause of the dermatitis. The eruption from this cause may be quite long in

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duration, compelling the workmen to suspend work either temporarily or permanently.

Blaschko has described the appearances of the dermatitis in aniline workers as of manifold character and of very quick onset often. He says the eruption is so typical that a diagnosis of the trade may be made from the appearances, the backs of the hands being much swollen, tinged here and there with dye, with intervening eczematous plaques. Hyperhidrosis of the palms (Grandhomme) has also been emphasized. The affection is often made worse by the means used for getting off the dye, chloride of sodium and chloride of lime, the use of the latter substances being partly responsible for the eczema.

Workers in Chemicals and Drugs, Arsenic, Chlorine.—Those who handle chemicals constantly are naturally subject to dermatitis of the hands from the irritation due to the contact. Chemists are very frequently annoyed by obstinate eczemas and are often obliged to suspend or abandon their occupation temporarily. Oftentimes an intense susceptibility is acquired—a state of anaphylaxis—to such a degree that a very small amount of exposure will call forth an outbreak. Sometimes chemists are very susceptible also to the fumes of various chemicals. The face is naturally affected very frequently from contact with the fumes. Fordyce has several times seen a similar anaphylactic condition from formalin in laboratory workers. In all of these cases the action of the chemical is undoubtedly increased by the constant immersion of the hands in liquids, and by the use of strong soaps and alkalies in the process of cleansing.

Arsenic, on account of its wide use in the arts, is a frequent cause of dermatitis. Preparations of arsenic have been used as pigments in the preparation of wall-papers, and of colored paper for other purposes, in printed and dyed cloths for garments and artificial flowers, in combination with aniline and other dyes, as preservatives of sizings and pastes, and for curing hides and bird skins. James C. White in his monograph on Dermatitis Venenata enumerates these various occupations in which arsenic is used, and states that the workmen engaged in manufacturing and those manipulating these products are subject to a great variety of inflammatory processes of the skin. An erythema is the first and mildest grade of inflammation produced by the action of arsenic on the skin, which is quickly followed by papules, vesicles and pustules. A further development is the formation of characteristic ulcers, round, of a grayish or reddish moist base, and sometimes surrounded by a dense induration. These may be quite deep and painful, and are commonly seated upon the hands, especially about the nails, and the forearms, parts which come directly into contact with the arsenic. The face, especially the regions of the lips and nose, behind the ears, about the neck, the scrotum and surfaces of the thighs adjoining, and the toes are other parts frequently affected. "Arsenic pock" is a name that has been given to arsenical ulcers of the hands in color workers. The dermatosis that frequently occurs on the hands of furriers, especially of those who are

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engaged in handling dyed furs of an inferior grade, has been attributed to the sulphide of arsenic that is used with lime in the curing of the fur. White has reported cases of arsenical dermatitis on the hands of taxidermists in whom the typical ulcerations about the nail were developed in a marked way. He records the case of a man who suffered from a severe dermatitis, so that the eyes were closed, and there was present considerable constitutional disturbance. It was found that he had been engaged for 3 days previous to the cutaneous appearances in fastening pieces of white webbing into sample books, the paper linings of which were covered with green arsenical pigment as determined by chemical analysis.

White says (Dermatitis Venenata, 1887): "Chlorine gas acts as an irritant upon the skin, producing prickling and redness when held in contact with it, and sometimes an eruption of papules and vesicles. Chlorinated soda and chlorinated lime solutions occasionally cause a mild degree of inflammation, erythematous and papular in character, in workmen employed in bleacheries, paper mills, etc."

Herxheimer described (1899) for the first time an affection which he called chlorine acne, which gave rise to numerous publications confirming his observations but disagreeing as to the etiology and nomenclature. It is observed in electrochemical establishments among workers in rooms in which free chlorine is obtained from chloride of potash by electrolytic decomposition. Hence it was concluded that this acne was caused by the chlorine, analogously to that produced by bromine and iodine, being breathed in and then excreted by the sebaceous glands. Doubt was thrown on this view later by the fact that a similar affection was not observed in establishments in which chlorine was generated by other than electrolytic means. Kaposi suggested that it was a tar acne, and this view, although at first rejected, has since attained much prominence, the direct agent being paranitrochlorbenzol which is generated in the electrolytic process.

The constant feature of this affection is the comedo, of the type common in acne vulgaris. At the start it is seen on the central portion of the face alone, but later it appears on the ears, neck, trunk and extremities, often in great numbers. With this comedo formation there is associated an eruption of acne lesions often attaining a large size, and presenting the appearance of sebaceous cysts. These lesions develop very rapidly, the affection reaching its full extent in a few weeks usually, and then remaining stationary for a long time, even if the patient has abandoned his work. Complete healing is the final result. Anatomically the lesions do not differ from those of ordinary acne and comedones. They differ clinically from these affections by their extension over the body generally. The affection is painless and without influence on the general health. It has seemed to be more common in summer than in winter.

Wood Workers.—A number of species of wood handled in the different trades have been shown to be poisonous to the skin of those working with

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them. These for the most part are the foreign species, satinwood, teakwood, ebonywood, etc. Epidemics of dermatitis from satinwood have been reported by Cash as occurring in the shipyards on the Clyde, and the East Indian satinwood is regarded as more irritating than the West Indian variety. The alkaloid chloroxylonine which the satinwood contains is regarded as the active agent, but Knowles found that the local application of East Indian satinwood dust caused the eruption in a person who had previously been poisoned by contact with chloroxylonine. The unprotected portions of the skin—the hands, wrists, face and ears—are the parts most frequently affected. Crocker has recorded cases of dermatitis in flute makers who use cocuswood, due, he thinks, to a resin in the wood, which belongs to the family euphorbiaceæ. Horand describes under the title "mains de crocodile" a dermatosis observed in workers in chestnutwood, which begins as an erythema and ends in a hyperkeratosis.

Flax and Linen Workers.—Purdon (quoted by White) described in 1875 a peculiar form of acne on the forearms of workers in the linen mills of Belfast, Ireland. The young girls who removed the bobbins from the machinery and the spinners were those chiefly attacked. The arms were the parts most affected; next, the face, owing, it was thought, to wiping the face with the oily hands. The eruption was of a papulo-pustular type, with a shotty feel in its early stages, and resembled smallpox somewhat.

Leloir in 1885 reported a skin affection in flax spinners in Lille. The eruption was situated mostly on the hands, and especially on the palms, more commonly the left one, and was peculiar to those who worked with damp linen. The hands of these workmen were kept constantly moist with the hot water from a trough through which the threads were made to pass for the purpose of cleansing the flax, and in this way the hands became covered with a mucilaginous coating, which was found to contain butyric and lactic acids combined with carbonate of lime in solution. The dermatitis was of an erythematous, vesicular and squamous type.

White observes that in the cases of Purdon and Leloir the poisonous action cannot be ascribed with certainty to the flax alone, as there are other irritating factors present. Thorough enquiries made by White of the proprietors and managers of flax mills failed to discover any irritating properties in the flax itself.

In 1889 Leloir described an epidemic of folliculitis and perifolliculitis in spinners. The place of prominence was the extensor surface of the lower limbs, especially where the wet trousers came into contact with the skin. It is rare that it affects the upper limbs as the workmen usually work without covering the arms, and it is characterized clinically by papules and papulo-pustules which may attain a considerable size. The pruritus may be quite severe. Leloir considers that the affection is due to the irritating oil with which the looms are impregnated. The location is explained by the soaking of the trousers in the oil and by wiping the hands on the trousers. The sub-

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jects affected were usually males, although some cases were observed in females when a thin closely fitting petticoat was worn. Leloir pointed out the aid that this dermatosis may be in legal medicine. He estimated that from 5 to 8 per cent. of the workmen were subject to this affection.

Knowles has seen outbreaks of dermatitis in five workmen in a cotton mill, four workmen in woolen mills, and four cases in weavers. He points out the fact that the dye houses connected with the mills are much more apt to be the seat of an outbreak than the mills proper.

White refers to an instance of a rash reported to him as occurring among workers in jute. There is also a German account of an eczema of the hands and arms prevalent in jute mills, attributed to the oil with which the fibers are saturated in order to work them.

Oliver has published photographs obtained from Dr. Glibert of Belgium, showing varying degrees of erosions on the palms of workers in flax, especially the female workers, due to the frequent contact of the hands with the irritating material contained in the liquid for spinning.

Tar, Paraffine and Petroleum Workers.—The various preparations of tar have been used from very early times quite extensively in the treatment of skin diseases. The indications for this group of preparations are in general the relief of pruritus and a diminution of inflammatory infiltration and hyperkeratosis—what has been termed a reducing action. Two forms of dermatosis may be produced by the pathological action of tar. One is a diffuse dermatitis, often accompanied by much burning and itching and which may merge into a long-continued eczema. The other form is an acniform inflammation of the follicles, the mouths of the follicles being often sharply defined by a black stain of dried tar, so that the appearance of comedones is simulated. Oftentimes these black comedo-like lesions are surrounded by an inflammatory border, and in this way the "tar acne" is produced. In some cases the comedo-like appearances are present without any inflammation of the follicles. This tar acne has sometimes been seen in workmen who are employed with machinery which has been lubricated with tarry compounds, and when the air of the workshop is filled with the vapor of tar. In some cases this tar acne changes into a more diffuse form of dermatitis, of an eczematous nature, which has been called "tar itch." In still other cases workers in coal-oil, and tar products generally, have developed indurated plaques and hyperkeratoses and multiple papillomata which have in turn degenerated into malignant growths.

White, writing in 1887, states that while it has been reported that workmen in petroleum suffer from a scarlatinoid eruption or from furuncles on exposed parts of the skin, the crude petroleum itself cannot have very irritating properties, as it has been used for years in considerable quantity for the destruction of scalp and pubic lice, without causing a dermatitis. He has seen teamsters, who were engaged in carrying the crude oil from the railroad tank to the refining factories, wash their faces and hands in a bucket of the oil. He

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thinks it probable that the poisoning arises from contact with some of the numerous products obtained by the various processes there employed, and not from the crude oil.

Lewin in 1888 published an elaborate article on general and cutaneous poisoning by petroleum. He made a trip to America and saw cases of skin affections in the Atlantic Refining Company, which had their seat especially on the upper leg, knee, and arms. There were often acniform lesions on the arms, as well as large furuncles with inflammatory appearances in the vicinity. He found that it was the heavier oils that caused the trouble, for no affections of the skin were found among those who worked with the lighter oils.

Derville and Guermonprez, writing in 1890, report that they examined between 400 and 500 workers in petroleum refineries, with the result that they found only four affected with papillomata, and these were the men who were engaged in cleaning the apparatus used in the last distillation of the petroleum. The clothing becomes saturated with the oily substance, and exhales a penetrating odor of petroleum. The forearms and hands are most frequently attacked, although other parts may be affected, such as the eyelid and scrotum. The warty growths are of a very variable size, and seated upon a deeply pigmented skin which is dry and often spotted with white cicatrices.

Mackenzie has described similar appearances in a man 30 years of age who worked in creosote. In this case the forearms and backs of the hands showed numerous comedones between which were warty elevations as large as a pea in some instances. There were similar lesions on the scrotum. The microscope showed epithelial degeneration.

Piffard quotes Ogston as describing a dermatitis of the hands, wrists, feet and legs in the workmen engaged in working crude paraffine. The eruption is composed of bright red nodules about the follicles, and these nodules gradually subside leaving the mouths of the follicles dilated and filled with black dots. The backs of the hands are most severely affected, and in exaggerated cases present a peculiar honeycombed appearance, due to thick clusters of distended hair follicles.

Florists, Gardeners, and Those who Handle Plants and Flowers.—At the time of the publication of his valuable monograph on Dermatitis Venenata in 1887, White estimated roughly that about 60 native plants or those introduced into the United States were capable in some way of injuring the skin. Since then he himself and others have added considerably to this list. Among the most common of the plants that call forth a reaction in the human skin are poison ivy, poison sumach, poison oak, the two species of nettle, Japanese primrose, etc. Gardeners and florists suffer frequently from a dermatitis of the hands and arms, extending often to the face and other parts of the body, from handling and working among these plants. A pretty serious acute dermatitis may be present, such as all are familiar with in intense

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cases of poisoning from ivy, extending in some cases over a large part of the body and causing great œdema and discomfort.

Lacquer is obtained from the juice of a species of rhus in Japan, closely allied to our native plant. White says that the history of the preparation of this celebrated varnish of Japan and its effect on the workmen engaged in its manufacture and use are very little known. Incisions are made in the bark extending about one-quarter of the trunk's circumference and just deep enough to reach the wood. The clear sap which flows out becomes rapidly darker on exposure to the air, and gradually takes on a dark brown almost black color. The sap is preserved in bamboo tubes, to keep it from turning black. It is shown by older writings that those who worked with this sap were frequently poisoned. White found that the embossed Japanese wall-papers that are used in our houses have produced severe inflammation on the hands of the hangers, in consequence, he assumes, of the abundant lacquer with which they are covered. Allen of Korea has reported that many foreigners as well as natives in the East are affected with "varnish poisoning," which is in some cases so pronounced that the person cannot pass a furniture shop where articles are being varnished without being poisoned. This susceptibility is more pronounced during the rainy season.

Citrus vulgaris, the bitter orange, is largely used for the manufacture of marmalade in Florida and in some parts of Europe. Bazin described the occurrence of a dermatitis of the face and upper extermities in workmen employed in peeling this fruit in France. The left hand, in which the fruit is held while being cut, is especially affected. In the United States it is stated that persons employed in manufacturing oil of orange peel suffer greatly from erythematous, papular, and vesicular eruptions of the skin, especially of the hands. White states that inquiry among the large orange growers of southern California fails to discover the occurrence of poisoning in those who handle the fruit. Marmalade is not commonly made there.

Vanilla comes from the bean of a plant cultivated in Mexico, Central America, the West Indies, and also in the East. It grows best in Mexico. White reports a case of acute dermatitis of the head and hands in a man who had been twice affected in the same way since he had been engaged in managing a vanilla commission house. In the manufacture of vanilla the beans are collected, put through a "sweating" process, turned frequently under blankets, and sometimes submitted to artificial heat, until they assume a dark chocolate or blackish color. In handling these pods many of the workmen acquire a dermatitis of the hands and face, which has been attributed to two causes: first, an acarus, which is improbable; second, in the lower qualities of pods it is known that an artificial method is used for coloring them black, and that this material is the oil of the cashew nut found in the rind, called cardol. White says that there is little doubt that these cases of so-called vanilla poisoning are from the cardol of this nut.

White was the first to call attention to the frequency with which those

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who handled the primula obconica, or Japanese primrose, acquired an artificial dermatitis upon the face and hands. Since then others have observed this occurrence, especially the English. This plant was introduced to European florists in 1882 from China. The first case observed was in a florist, who was attacked at the same time during 2 successive years, a day or two after this plant came into bloom, and on each occasion two of his assistants were also affected. A botanist finds that the hairs with which the leaf and flower stems are abundantly provided are easily detached, pointed, jointed, and contain a large amount of silex, and that none of the other species of primula are provided with these hairs; hence it is probable that they produce the inflammation.

Walsh has described a dermatitis among flower pickers in the Scilly Islands, the so-called "lily rash." The narcissus is cultivated in great quantities in these islands and exported in the spring. The workmen who cut the flowers are subject to an artificial dermatitis of the hands, arms, and face; not all varieties are said to be equally harmful, the most dangerous being the "campanelle."

An epidemic of dermatitis among the workers in rice fields has been alluded to by Mantegazza, affecting the feet, legs, hands and arms. He found that it was only in certain fields and during certain years that this occurred, and attributed it to a small plant, covered with numerous spines up to the top, which he found in the fields in question. These spines possess no secretory apparatus that produces a toxin, but have a mechanical action, boring into the skin of the laborer who goes into the water and therefore rubs against the leaves of the plant.

Quinine and its compounds are capable of setting up a severe dermatitis in those handling them. White was not able to find any history of the occurrence of inflammation of the skin from contact with the crude cinchona bark or other parts of the tree. Bazin stated that the workmen employed in the principal quinine factories of France, Germany and England were liable to a cutaneous disturbance, which was sometimes so severe as to cause them to give up their occupation. These accidents are most common in Germany and France. The workmen affected are chiefly those who boil the bark, those who convert the quinine into the sulphate, and those who bottle the sulphate. A simple residence in the factories, without being employed, is sometimes sufficient to cause this affection. The affection had the form of a typical, acute artificial dermatitis and was due, it was thought, to quinine emanations. All of the workmen did not seem to be equally predisposed to the affection, as some had been engaged in this occupation for years with no ill effect. In some cases the subjects seemed to become habituated to these influences, and the attacks gradually ceased; others were forced to give up their work permanently. White found that occasional cases occurred among the workmen in American manufactories. Idiosyncrasy seems to play a more prominent part in quinine rashes than in most others.

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Musicians.—Stern has divided the dermatoses of musicians into three categories: 1. A sycosis of violinists. He collected six cases, three of whom were musicians, one a teacher, and two tradesmen, in which a sycosiform eruption developed on the left half of the face, at the lower boundary of the beard, in essentially the submaxillary region, the part of the skin against which the violin is accustomed to press in playing on that instrument. 2. An eczema of flute players, a moist dermatitis of the lips and neighboring parts of the cheeks and neck. In one case the eczema recurred every time the flute playing was resumed. An instrument maker to whom the flute was submitted said that it was made of "Grenadillholz," red ebony-wood from the East Indies, and declared that he had received back a similar flute from a musician because it had caused a similar lip affection. 3. Lip affections of trumpeters. In a number of cases on the lower lip near the middle a sort of abortive furuncle occurs, without tenderness. In other cases he observed scaling, horny excrescences in the same class of people, appearances much like those of cornu cutaneum.

Physicians, Surgeons, Nurses, Etc.—Since the adoption of the principle of asepsis, the use of various solutions for the treatment of the skin both of patients and of surgeons, dressers, nurses and attendants has developed many cases of obstinate dermatitis, especially upon the hands. The constant use of soap, water, corrosive sublimate, carbolic acid, strong alkaline solutions, creolin, formalin, etc., is responsible in great measure for these conditions. The same conditions are responsible for a similar occurrence in dentists. Several instances have come under the writer's notice in which surgeons, after repeated rests from all irritating work, have been compelled finally to abandon their calling on account of the lamentable and dangerous condition that their hands were continually in. Numerous instances have also come under the writer's knowledge in nurses who have been obliged to give up their training and seek some other occupation. Formalin is a very irritating substance, and the cause of many cases of dermatitis. In some instances even a slight exposure to the fumes will be sufficient to cause an irritation of the skin. This susceptibility often increases after several attacks.

In this connection it will suffice merely to mention the possibilities that are offered to those attending the sick of becoming infected with various diseases. The numerous cases of physicians who have become infected with syphilis through a wound in the finger, often in the practice of obstetrics and gynecology, are well known. The form of tuberculosis of the skin that has been called tuberculosis verrucosa cutis, and that is due to direct inoculation of tuberculous virus on the cutaneous tissues, is seen occasionally in physicians and nurses, as well as in medical students and dissectors, ward tenders, etc.

Butchers, Cooks, and Those who have to do with Animals and Animal Products.—Most of the cutaneous affections caused or influenced by special occupations, which have been considered in the preceding pages, are a serious

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menace to life only in exceptional instances and, as a rule, when they become generalized. Among people who deal with animals and with animal products we find a class of cases due to direct infections that are, as a rule, much more serious in their results and often a menace to the life of the individual.

Pyogenic and Tricophytinic Infections.—Very numerous instances of the pyogenic affections of the skin, such as impetigo contagiosa, furuncles, carbuncles, etc., are found in butchers, cattlemen and those who have to do with dead animal matter. The opportunity for acquiring these affections in the course of such occupations is obvious. Tinea tricophytina and allied forms of vegetable parasites often are found in animals, especially domestic animals. Frequent instances of contagion from cats and dogs are found in children, and occasionally, as is natural, these affections are transmitted to dog fanciers and those who have the care of such animals. Tinea tricophytina has been described as occurring in nursemaids and in those who take care of similarly affected children in institutions and day nurseries. Several years ago a herder from Vermont was received into the skin ward of the Massachusetts General Hospital suffering from a very aggravated case of tinea of the beard, so-called parasitic sycosis. The skin of the maxillary regions was much tumified and indurated and covered with large nodular sycosiform tumors. It was ascertained that others among his fellow-workers were affected in a milder degree with the same trouble, and that there had been an epidemic among the cattle of a cutaneous disturbance causing a falling of the hair.

A much more serious cutaneous infection may occur in butchers, an example of which was observed by the writer in 1902. The subject was a wholesale meat cutter, 28 years of age, who was affected with a universal pemphigoid eruption accompanied by fever and severe constitutional symptoms, following a cut received on his hand in the course of his occupation that had become septic. This patient recovered, but Pernet, who has made a special study of this subject, finds the prognosis very grave, most cases ending fatally. He collected 16 cases, one-half of which were in butchers; but in all of the remaining cases the occupation was one that brought the subject into contact with animals or animal products, one case being that of a tanner, another that of a person who had milked some cows that had a bullous affection of the udders and teats. Pernet's conclusions were as follows: 1. That there is a group of rare cases of acute bullous eruption, accompanied by severe constitutional symptoms and generally ending fatally, which affects butchers. 2. The disease follows a wound on the hands or fingers. 3. It is probably due to a micro-organism. 4. The same infectious disease probably occurs in other persons who are brought into contact with animals or animal products. Allen has reported a case in a blacksmith.

The writer's case occurred simultaneously with the prevalence of an epizootic of "foot and mouth" disease in New England, and the possibility of a relationship between the two affections was considered. With regard to the

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occurrence of foot and mouth disease in man, while strictly medical text-books and publications contain little on this subject, there are many allusions to it in veterinary literature. According to Nocard and Leclanche, Segar in 1765 was the first to report cases of the infection of man by animals from this source. Nicolaier, in Ebstein and Schwalbe's Handbuch der praktischen Medizin, describes foot and mouth disease when occurring in man as characterized by fever, constitutional symptoms, and by a generalized eruption of vesicles and pustules, especially marked on the fingers and feet, and affecting the mucous membranes of the lips, cheeks and tongue, when the poison has gained entrance in this way. He says that when the infection has taken place through an abrasion of the skin, as is often the case in milking or in slaughtering diseased animals, the eruption appears first at the place where the poison entered.

Anthrax, *glanders*, and *actinomycosis* are among the affections that may occur in animals and be transmitted to man. Anthrax is very common, especially in cattle and sheep, in various parts of Europe and Asia. The disease in man is most common in those who have to do with animals or animal products, such as slaughterers, farmers, tanners, wool-sorters, stevedores, meat inspectors, employees in brush factories, etc. It is said to be transmitted by indirect infection more often than by direct, often by means of flies, or from the hair, hides, wool, etc., of animals that have died of the disease. As is well known, it is caused by the bacillus anthracis, one of the earliest pathological microbes discovered, and in its external, most common form is characterized at the point of inoculation by a pustule, which rapidly changes into a central gangrenous area surrounded by a brawny swelling covered with small vesicles or pustules. By the time the central necrosis has taken place, severe constitutional symptoms have usually set in. Ravenel, quoted by Stelwagon, has described an epidemic of anthrax in tanneries near Philadelphia, in the course of which 12 men and 60 head of cattle died during the course of a year, the men being employed in the tannery as operatives and the cattle fed in pastures through which ran a stream polluted with the refuse of the tanneries.

Glanders is usually contracted from horses and is seen therefore most frequently in stablemen and in those who have to do with these animals. It may be acquired through a break or lesion of the skin or through the mucous membrane of the mouth or nose. In some instances the mucous membranes are the parts most affected; in others large nodules, the so-called "farcy-buds," may arise from infection of the lymph glands, and there may be also a thickening of the lymphatic vessels. The skin eruption is in general of a pustular nature, but is not especially characteristic and is often not especially prominent and even wanting. The nasal discharge may be very profuse or quite moderate and is due to ulceration of the mucous membrane, which may penetrate to the bone. A chronic form has been described and its description and history elaborated by Besnier. The constitutional symptoms are usually pronounced and the mortality is high. The disease is usually contracted

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either by direct inoculation of the secretions on a wound or through the mucous membrane, from the snorting of the animal affected. Crocker quotes the case of a laundress who was infected from washing the clothes of a coachman who had died of the disease.

Actinomycosis does not in most instances affect the skin, the internal organs being the parts most frequently involved. It has been estimated that the disease is located in the skin in only about 3 per cent. of the cases and, although sometimes primary in this situation, it is usually secondary to internal involvement. The seat is usually about the face, jaw and neck, and it has often been proved to have obtained access to the jaw through a decayed tooth. The clinical appearances are those of hard nodular swellings, which develop fistulæ discharging pus mingled with which are the characteristic yellow granules of the ray fungus. This ray fungus is the cause of the affection and is supposed, although it has not been proved, to flourish on hay, corn and straw. At all events, the affection is undoubtedly caused by inoculation of the ray fungus, and is most commonly seen in those who have to do with cattle and horses, including farmers, dairymen and millers. It has been thought to have been acquired by chewing straw, grain and malt, a common practice among stablemen, farmers, dairymen, etc., the infection probably occurring by inoculation through a carious tooth.

Tuberculosis in the form of tuberculosis verrucosa, that has been referred to as occurring among physicians, dissectors, nurses, etc., is also very commonly seen in butchers, cooks, cattlemen and those who have to do with animals and animal products. It is due to direct inoculation of the skin with the tubercle bacillus and is the most frequent type of cutaneous lesion due to this cause. The lesion has also been called the anatomical wart, verruca necrogenica, etc., from the fact that it was first observed on the hands of anatomists and those who made autopsies, at a time when its tubercular nature was unsuspected. It is seated on the hands and arms almost exclusively and presents the appearance of a papillomatous nodule with a rather soft, infiltrated base. It may easily assume a more pustular appearance from secondary infection. This form of tuberculosis is very slow in its course, and the patches may remain for years without undergoing much change. In some cases, however, it may result in infection of the neighboring lymphatics and in foci of lupus in the vicinity. A disseminated tuberculosis and an infection of the internal organs, with fatal termination, may also follow occasionally. This form of tuberculosis has been seen not infrequently in those engaged in caring for tuberculous patients, especially relatives, where the rules of hygiene, so rigorous in all well-conducted hospitals, are not adhered to.

Erysipeloid is the name given to an infection, usually comparatively mild in character, that is acquired by butchers, fish dealers, poultry dealers, and cooks in the pursuit of their occupations. Of the cases seen by the writer, in Boston and vicinity, the majority were in people who handled fish in some way, either as fish dealers, fishermen, or cooks. It is usually situated on the

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fingers and hands and appears as a sharply bounded, spreading erythema, not unlike a mild form of erysipelas, and tends to spontaneous disappearance after a few weeks, although its course may be much abridged by treatment. Rosenbach has discovered a micrococcus of the order Cladothrix found in decomposing animal matter, which has produced the affection experimentally when inoculated in pure culture. He obtained a pure culture on gelatine from a case of erysipeloid acquired from foul cheese, which he inoculated upon his own arm in three places. Three days later a raised erysipelatous patch appeared at the site of inoculation, which spread peripherally. Gilchrist has recorded 329 cases, of which 323 were caused by crab bites or lesions produced by crabs. The cases were naturally chiefly in those who dealt in crabs or were engaged in cleaning them. Gilchrist and others in Baltimore have totally failed in their attempts to find a causative organism, nor were they able to produce the affection by experimental inoculation.

Miscellaneous Occupations.—Under this heading it will be convenient to group a few observations of less prominence and importance than those that have been discussed. The *theatrical profession* has been the subject of an article by Brown entitled "The Theatrical Profession as a Factor in the Dissemination of Disease." Scabies, pediculosis, syphilis, and other contagious cutaneous affections are common among theatrical troupes, as the opportunities for communicating such diseases are considerable. The mode of life, sojourn in cheap hotels, frequent changes of domicile, the use of cosmetics, and above all the danger of infection from costumes and wigs render such affections more frequent than usual. The writer has had under his care a celebrated actress who suffered from a very serious parasitic affection of the scalp, attended by almost complete loss of hair, which she had acquired from wigs used in the theatre.

Since the introduction of the X-ray for diagnostic and therapeutic purposes, very severe and sometimes serious conditions of the skin have occurred in operators who had been engaged in this work for long periods of time; a persistent, unusually rebellious dermatitis, and a condition of atrophy, telangiectasis and keratosis, resembling the lesions seen in the rare affection called xeroderma pigmentosum and often followed by cancer, are not infrequently the result of this occupation. This subject is fully treated in the chapter on Cancer.

Audry has published an observation on a papular and suppurative lesion in *milkmen* which affects the back of the hand, causing it to become greatly inflamed, and he thinks this is analogous to the cases published by Winternitz of nodular lesions on the hands, forearms and noses of milkmaids.

A disease of the nail has been described by Glibert of Belgium in *furriers*, especially those who scrape rabbit and hare skins in the manufacture of artificial furs. The work of separating the aponeurosis that occurs on the undersurface of the hide causes the groove under the nail to become deeper, and the nail is subsequently detached from the finger and falls off. The thumb and

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the middle and ring fingers of the right hand are especially affected. This has been said to be quite common in the fur pullers of Belgium. A keratosis formation of the palms and flexor side of the fingers has been observed in those employed in removing the hair from hogs. Stein has seen a similar appearance in glass workers. Spietschka reports a peculiar affection of the backs of the hands and fingers in young women workers in *enamel* manufactories, consisting of nodular elevations with infiltrated edges, hollowed at the top.

Under the title "*grain itch*" Schamberg has described an affection that became quite persistent in Philadelphia in the spring and summer of 1909. It was characterized by an urticarial eruption, the wheals in many instances exhibiting at their summits a central pinpoint-sized vesicle. This eruption was usually accompanied by most intolerable itching, which was worse at night, and mild fever. This affection was found to be especially prevalent among farmers and laborers who handle sacks of wheat, barley, and other grains, and to be due to a mite, the Pediculoides ventricosus, found in grain or straw.

TREATMENT

It is impossible to do full justice to the treatment of the various professional dermatoses without discussing at some length the methods of treatment applicable to a large number of morbid cutaneous conditions very varying in character. To do this would be to exceed the limits or purpose of this article. With regard to prophylactic treatment (the most important part of the therapeutics of occupational diseases), the avoidance of the exciting cause is naturally the first matter that presents itself. Total abandonment of the occupation that has proved injurious is not infrequently necessary. In many instances less drastic measures may be sufficient, and these measures naturally vary according to the specific occupation and often according to the individual case. In the case of people who are obliged to have their hands in contact with water and soap quite frequently, such as housemaids, bartenders, etc., the use of rubber gloves has become a valuable prophylactic aid, now that these articles are manufactured so well as to withstand moderately rough usage. In these cases of susceptibility to irritation from soap and water, as well as in all other cases of poisonous irritants, the application of a bland, softening and protective cream after exposure, or before going to bed, is of much value. Either simple cold cream ointment or a combination of cold cream with a small proportion of lanolin will be found most serviceable. Loose white cotton gloves worn during the night are a good substitute for a protective bandage. In the cases of nurses, physicians and those whose hands are exposed to contact with strong antiseptics, thorough anointing with a bland cream in this manner is most useful as a preventive. These precautions should be strictly followed as soon as the subject shows a well-defined idiosyncrasy to any form of irritation. Various

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measures of protection have been devised to suit the particular kind of irritation or the especial work of the individual. Masks have been used in various occupations in which the fumes that are developed have proved poisonous. Measures of this nature have been employed by those working with lacquer.

The immediate treatment of the greater part of the cases of occupational dermatoses resolves itself into an understanding of the principles of treatment of dermatitis generally, since a dermatitis either of the type of dermatitis venenata or of the eczematoid variety is most frequently met with. An acute dermatitis venenata demands first of all the removal from the skin of any of the irritant that may still be adherent. For this purpose soap and water is often essential, although, as has already been seen, these agents are irritating in themselves, and their vigorous use is often responsible for the aggravation of the trouble. Bran or starch may be added to the water for the purpose of "softening" it, as in the case of infants and those with delicate skins, and the various superfatted soaps, less irritating on account of the excess of fat that they contain, may be employed. The use of alcohol and strong alkalies for the purpose of removing stains and obstinately adherent substances should be resorted to as sparingly as possible.

It is needless to point out the fact that there is no form of internal treatment that can be regarded as in any way specific in the management of dermatitis. Each case must be treated for itself. The condition of anæmia that is often seen in factory operatives, and that has been especially referred to in the case of furniture polishers, requires careful attention, and the same may be said as to dietary errors, and especially the excessive use of alcohol. The wise adviser will pay careful attention to the correction of hygienic conditions as far as possible, and to the patient's general well-being.

Locally, in the acute forms, lotions mildly alkaline and slightly carbolized, with or without a bland soothing powder suspended in them, and perhaps with the addition of a small amount of glycerine, are of great value at the outset on account of their antipruritic properties, as well as their relief of the acute inflammation. Where there is not a large amount of crusting, a bland dusting powder of talcum, zinc, or starch, is a useful application. Crusts, if thick, should be softened off by applications of oil, or by moist compresses continually worn. At a somewhat later period, or if the early stage is absent, bland ointments and pastes are indicated, in which the parts affected are wrapped, with an outer covering of soft cotton or linen. In a large majority of cases of dermatitis from external irritants these measures are sufficient to control the individual attack at least, provided the source of irritation is removed. The more chronic forms of eczema are, however, well represented among the occupational dermatoses. In these cases the more stimulating applications are to be resorted to on the same principles and with the same regard for the individual case as in eczemas with a different etiology.

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As regards the more serious affections, such as those acquired in handling animals and animal products, in the course of medical and surgical attendance, etc., the greatest care must naturally be taken to prevent the entrance into the skin of any foreign matter. On this account people engaged in these occupations should be instructed as to the dangers attendant upon their work, the probable modes of inoculation, and the importance of protecting all wounds or abrasions of the skin by some form of protective dressing.

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CHAPTER II

CANCER AND OCCUPATION. X-RAY, RADIUM

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1. CANCER

Definition.—The term cancer may be used in two senses: in its general application it signifies any form of malignant tumor; in its restricted and more accurate sense it is synonymous with carcinoma, a malignant epithelial tumor. A tumor is a mass of tissue which arises from the atypical proliferation of certain tissues of the body and which is without function. Tumors which possess powers of unlimited growth, which invade and interfere with the activity of normal tissues or organs, which penetrate the circulatory system and thus become disseminated throughout the body so that death ultimately results are termed malignant. Tumors which grow very slowly by expansion or become stationary, and neither tend to invade normal tissues nor become disseminated so that they are not ordinarily fatal, are termed benign.

Incidence.—The frequency of cancer is shown by the fact that it ranks fifth in the list of causes of death.[1] According to the mortality statistics of England for the year 1909, about one in 10 males and one in 7 females who died at the age of 35 years or over succumbed to cancer.[2] This condition differs from many other diseases in that it is almost invariably fatal if not artificially eradicated. The cases recorded of spontaneous recovery from cancer[3] are extremely rare, so that the prognosis of untreated cases is bad.

Distribution.—Cancer has a wide zoölogical distribution, occurring in many different species, in fact in representatives of practically all orders of the vertebrates. The greater number have been found in man and domestic animals or in animals living in captivity. Tumors have been reported, however, in animals living in the wild state under natural conditions so that, notwithstanding the meagerness of such observations, it is certain that the development of tumors is not restricted to the artificial conditions of domestication.[4, 5] The swellings which accompany various parasitic infections in lower animals are often reported as tumors, but the term tumor is in such instances used loosely to designate any swelling irrespective of its character. The frequency of tumors varies in different species, and some appear to be practically free from any form of malignant growth. The longevity of the individuals in each species should be taken into account, however, in determining relative frequency. Thus, for example, tumors appear to occur fre-

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quently in short-lived animals, such as the mouse and the rat, which live no more than 2 or 3 years at the most. Although statements based on statistical studies or upon personal impressions would tend to show that certain less civilized races are relatively exempt from cancer,[6,7] such conclusions cannot be accepted until more systematic clinical and pathological investigations are carried out among wild tribes. It frequently requires only systematic search for a condition to be revealed where it had not previously been suspected.

Age.—The incidence of cancer increases with age, especially with respect to involution changes in certain organs. If the incidence is represented by a curve, we have in early life the deaths from congenital tumors, following which there is a drop and the curve remains low during puberty, rises rapidly toward the end of the reproductive period and with advanced life tends again to fall. Age then is of considerable importance in the development of tumors. In this respect there is marked variance from the definitely infectious diseases which either occur early in life or are more or less distributed with respect to age incidence. The changes which take place in the skin and their bearing on the development of skin cancer is especially important.

Theories.—Many theories have been advanced in explanation of the etiology of cancer. It will be impossible to discuss all these theories within the allotted space, and thus only a number of the more important will be reviewed. Thiersch and Waldeyer[8] have presented the hypothesis that there exists between the epithelium and connective tissue an equilibrium through which the growth of the former is held in check. On the degeneration of the connective tissue in old age the epithelium proliferates and tumors develop according to this hypothesis. Cohnheim[9] attached great importance to the displacement of cells or groups of cells from their normal relations during the course of development, the so-called "embryonic rest" theory. This idea of the dislocation of cells has also been utilized by Ribbert[10] in explaining the origin of tumors. He, however, considered other forms of tissue dislocation than that which occurs naturally in development. He found that epithelium implanted in the connective tissue remained alive for a time and was capable of a certain amount of growth. Although epithelial cysts could be formed in this manner, the transplanted tissue possessed only limited powers of growth and never resulted in a tumor. Ribbert utilized the disturbance of the normal relationship of tissues in chronic inflammatory conditions to account for the origin of tumors.

The theory of the gametoid nature of cancer tissue reached its greatest development in articles by Farmer, Moore, and Walker[11] although it is not original with them. By this theory the unusual activity and growth of tumor tissue is considered to be due to a process of fertilization taking place between cells of the somatic tissues. The fusion of such cells gives rise to a sort of hybrid tissue capable of active growth. A support for this theory was claimed in the presence of a reduced number of chromosomes in dividing

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cancer cells, also in the morphological similarity of the chromosomes to those which are found in the reproductive cells. Von Hansemann[12] explains this reduction in the number of chromosomes as the result of asymmetrical mitotic division and suggests that with the loss of chromatin there may be also a loss of differentiation in tissues, which he terms "anaplasia."

The view that cancer develops as the result of some specific infection has found many adherents and through the employment of a great variety of methods many different organisms have been reported as the cause of cancer. None of these has found acceptance although the association of certain parasites with inflammatory conditions which may be followed by cancer is worthy of further consideration. In a single species, the common fowl, it has been demonstrated that tumors may be produced by the introduction into normal fowls of an agent which is readily passed through a Berkfeld filter and which also resists drying sufficient to kill all tumor cells.[13] Whether this is of the nature of a microörganism or of a chemical substance is not yet conclusively shown. Repeated attempts to isolate such an agent from mammalian tumors have invariably failed.

In the etiology of cancer the importance of certain agents, which produce long-continued injury and chronic inflammation, is now universally recognized. Such agents demand consideration from the standpoint of occupational disease and will thus be discussed somewhat in detail later on.

The Question of Contagion.—The question is frequently raised as to the possible contagiousness or infectiousness of cancer. It is definitely known that cancer may be transplanted from one portion of the body to another. This frequently happens naturally during the course of the disease either by the apposition of a tumor to the surface of healthy tissue or by the transplantation of tumor cells loosened in the fluids of the body, and it may occur artificially during surgical operation. Furthermore it would probably be possible, at least in some instances, to transplant tumor cells to normal individuals, since this is actually done in the experimental propagation of tumors in lower animals. The danger of the transplantation of tumor cells from tumor patients to normal individuals is undoubtedly very slight, and is theoretically possible only in instances where living tumor is brought into contact with abrasions or introduced by accident through incision or puncture. There is, however, no instance recorded of the accidental transplantation of human tumor to normal individuals either during surgical operation or pathological examination. If there were an infectious agent concerned, we would expect the nurse who is in attendance and the surgeon who is frequently operating on cancer cases and especially the pathologist who is brought into frequent contact with tumor tissue to show a high incidence of cancer. This, however, has never been demonstrated.

The Question of House Infection.—The occurrence of a number of cases in a single dwelling or in a certain locality has raised the question of house infection.[14] It has been pointed out, however, that the so-called "cancer houses"

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are usually occupied by individuals of more than the average age of the community at large and are also explained in some instances by cheap rentals with a consequent tendency of people with chronic disease to gravitate toward such localities. There is furthermore no evidence to indicate that the attendants in certain hospitals, almshouses, and other institutions where such cases are cared for, show a higher incidence than others. When the frequency of cancer in a community at large is taken into account, it would be expected that by mere chance distribution a large number of cases would occasionally be grouped in a restricted locality or in a single dwelling.

Heredity.—In the consideration of the part played by heredity in the occurrence of tumors, it has become more or less of a dogma in medical instruction that heredity is here of no significance. This teaching is based upon the investigations of Pearson[15] and others and was made on the basis of the theory of inheritance which has never been successfully applied as a working hypothesis and which is not recognized at the present time. Sufficient data have been collected in the lower animals to reopen this question, so that the subjects of heredity with reference to cancer in human beings is now also under investigation.[16, 17, 18] Tumors of a similar nature occur in closely related individuals sufficiently often as to create the belief not only among the laity but also in the minds of many physicians that heredity is of importance. While the data collected up to the present time from animal experimentation is insufficient to serve as a basis for any sweeping conclusions concerning human inheritance, we are of the opinion that the results already obtained are sufficient to call for a reconsideration of the subject of heredity with reference to cancer.

Hyperplasia.—While certain tumors appear to rise *de novo* without the knowledge of any preceding abnormal condition, many are preceded by conditions which are of long standing and which are frequently spoken of as "precancerous." There are various agents which produce unusual growth or proliferation of tissues. Such tissues during proliferation may correspond more or less closely in structure with the normal tissues. An increase in the amount of the tissue as the result of proliferation is termed hyperplasia. The increases in the size of the tonsils under conditions frequently met with in early life are examples of such hyperplasia. Certain hyperplasias are attributable to known causes such as microörganisms or higher parasites, others are of unknown etiology, and we also have examples of the compensatory hyperplasia to supply an increase in function by reason of loss of parts or other failure elsewhere.

During hyperplasia there is frequently a loss of differentiation of tissues which von Hansemann has termed "anaplasia." In fact, it appears that structural differentiation may bear an inverse relation to the rapidity of tissue growth.

Metaplasia.—Under certain stimuli certain tissues may not only depart markedly from the normal but become transformed to quite another type and this is known as metaplasia. The transformation of the glandular endome-

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trium into stratified squamous epithelium as the result of eversion of the uterus has been considered an example of such metaplasia, but here there is the possibility that the transformation is due to replacement with squamous epithelium growing in from contiguous surfaces rather than true metaplasia. A better example is to be found in the transformation of the epithelium of the bile ducts as the result of the presence of parasitic worms. This organ is completely isolated from all surfaces covered with squamous epithelium and may therefore be taken as a true example of metaplasia. In certain forms of

FIG. 17.—Hyperplasia in vegetable tissues due to infection with a myxmycete, *Plasmodiophora brassicæ*. Several of the slendor normal roots of the cabbage are apparent but nearly all are greatly thickened as the result of proliferation.

chronic pancreatitis, the glandular portion of the organ may become greatly modified and resemble cancer. The bronchial epithelium may also become changed from a columnar to a squamous type in certain instances.[19] Such conditions occur naturally but some may also be produced experimentally by infecting animals with the proper parasites. These changes, however, are not to be regarded as constituting true tumor growth although they may closely simulate the latter. They differ in this respect that they are self-limited and have a tendency to return to the normal especially when the exciting cause is removed.

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FIG. 18.—Metaplasia of the epithelium of the biliary ducts in fiber zibethecus as the result of the presence of a species of trematode, one of which is shown in longitudinal section.

FIG. 19.—Irregular proliferation simulating adeno-carcinoma in chronic pancreatitis of the cat.

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Injuries and Chronic Inflammation as a Basis for Cancer

There is now available a large amount of data which shows that long-continued inflammation due to various causes is frequently followed by, in fact paves the way for, the development of cancer. Probably the most convincing example is the production of cancer following long or repeated exposure to the X-ray, an unusual and artificial agent which will be referred to later on in more detail. A considerable number of tumor cases

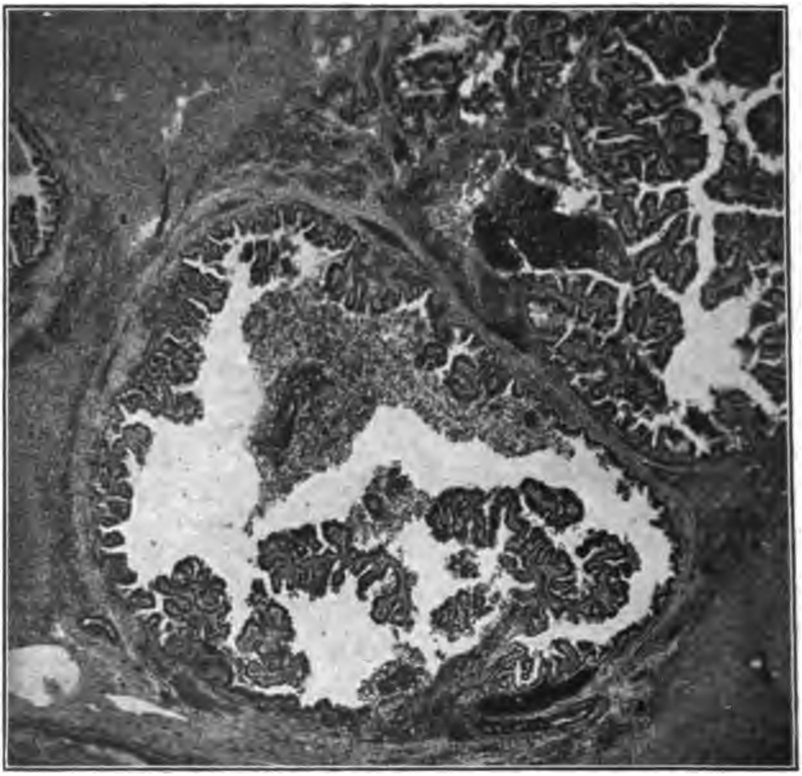

FIG. 20.—Coccidium nodule of the rabbit's liver simulating papillary adenocystoma. This lesion differs from a tumor in that it is self limited and, following the elimination of the causal agent, becomes replaced by scar tissue.

give a history of trauma preceding the appearance of tumor. In this connection it is necessary to take into account the tendency of human nature to find a plausible cause to which to attribute any infirmity. We should also consider the undue prominence given certain injuries on account of the previous involvement of certain structures by tumor. Thus an individual attributes the development of sarcoma of a given bone to a blow or other injury although it may very well be that no great importance would have been attached to such an injury in case the bone had

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been normal at the time. From a medico-legal aspect such cases present most perplexing problems. It is possible, even probable, that in a limited number of cases, trauma is to be considered as a contributory cause. The principle cause would appear to lie in the peculiarity of the individual, for trauma is of so great frequency that it cannot in any sense be regarded as the specific cause of any type of tumor. The involvement of any portion of the body by the metastatic growth of tumor previous to a given injury is usually more easily determinable either by the clinical evidence of tumor elsewhere, by the histological character of the tumor, or by post-mortem demonstration of the primary tumor located elsewhere. The frequency of the fracture of bones secondarily invaded by tumor which has originated elsewhere is familiar. A radiograph taken as soon as possible after the fracture should usually show and serve as a record of the presence of either primary or metastatic growth.

The number of agents that are known to produce chronic inflammation of such character that new growth frequently follows, is continually growing and it is with certain of these that we are concerned in the consideration of occupational disease. There are agents to which all individuals are exposed to a greater or less degree that are mildly injurious. The undue or prolonged exposure to the skin to such agents evidently has a tendency to bring on a premature senility of its tissues especially in certain individuals. The effects of light, heat, and drying on the exposed portion of the skin are familiar to medical men and it is upon these surfaces, especially on the face and hands, that such changes appear in later life. The more immediate effects of undue heat and cold on the skin are well known, especially to dermatologists, and the future condition of the tissues will depend on the powers of repair in each individual case. For example, by the habitual application of cold, a dry scaly condition of the skin may be produced at will in many individuals. Thus abnormalities in the form of undue greasiness and accumulations of the cornified epithelium, the so-called keratosis senilis, are found on the exposed portions of the skin of the aged in addition to a more generalized tendency to atrophy and loss of hair and glands. Certain individuals possess a pronounced idiosyncrasy to light, for example, and light has been shown to be unusually injurious to individuals subject to a skin affection known as Xeroderma pigmentosum.[20]

There are certain specific diseases that produce chronic inflammation which is not infrequently followed by cancer. Thus the long-continued injury to the skin in Lupus vulgaris serves as a basis for the subsequent development of cancer. Cancer may also develop in chronic syphilitic lesions, and that there is no incompatibility for the coexistence of these various diseases is shown by the occurrence of all three in a single patient.[21] In order to ascribe etiological significance to these conditions, it should be shown statistically that skin cancer is more frequent in such cases than in an equal number of the population at large of comparable age. The infection of the pelvic

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veins with a parasitic worm, Schistosomum hematobium (Bilharzia), and the inflammation resulting from the passage of the ova of these worms through the bladder wall is said to be frequently followed by carcinoma of the bladder. Since this infection is confined to certain regions of the world, in order to prove this point it should likewise be shown that cancer of the bladder is more frequent in such regions than elsewhere. As a matter of fact, statistics are so fragmentary and statements so conflicting with regard to this point that it is impossible to draw any conclusions. It seems probable that an undue amount of granulation tissue and hyperplasia of the mucosa of the bladder has given rise to this view and that with the removal of the cause such conditions would be ameliorated instead of progressing to a fatal issue as is almost invariably the case with true carcinoma. Cancer is occasionally reported accompanying infection with unusual parasites. For example, carcinoma of the bile passages or liver has been noted in infection of human beings with a species of fluke which is ordinarily found in the cat.[22]

Habits.—Of special interest is the influence of habit on the frequency of tumors. The use of alcohol in large amounts since it is commonly attended with more or less exposure or disease, is probably of some importance in bringing on a premature senility of the tissues. Likewise the tobacco habit may be attended with long-continued injury to portions of the mucous membrane. Countries in which preparations of betal nut and lime are habitually used for chewing purposes show a high incidence of cheek carcinoma.[23] Statistics for such countries are especially interesting in that they show a high frequency of carcinoma of the cheek in women, whereas new growth in this location is infrequent in women of European nations. Cancer of the abdominal wall is relatively infrequent in most countries, but is frequent in this location in certain parts of India where the body is warmed artificially by a small stove, the Kangri.[24]

Summary.—The effects of the various agents enumerated should be regarded in various occupations. Those which involve excessive exposure to light, heat, and cold may be expected to be of influence in bringing on a premature aging of the exposed portions of the skin and thus to factor in the development of skin cancer. Since, however, most of the tumors of this type are of comparatively slight malignancy, such influences are not of first importance. Occupations such as that of the soldier which expose individuals to certain parasitic infections, such as Schistosomiasis, etc., must be taken into account. The occupation of the physician and that of the nurse, however, are not shown to be dangerous with respect to direct infection from cancer cases. No very marked preponderance of cancer in any of the more usual occupations is shown by Newsholme's statistics.[25] The incidence of cancer in chimney sweeps, however, is more than double than in any of the other occupations. Since these statistics are based upon data relating to cancer in general, it is not remarkable that they show no more. If the statistics were collected for each type of tumor much more significant results

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would be expected with reference to the occupation. Notwithstanding this there is a strikingly high incidence in chimney sweeps.

Special Occupations.—The distribution and character of the tumors and the preceding conditions which are associated with certain occupations are of special interest. The occupations which have to do with the manufacture or use of certain coal-tar products are to be especially considered. In such occupations the exposure of skin surfaces to the irritating substances is said to be followed by a more or less characteristic type of inflammation or by warty growths. The liability to tumors under such circumstances is recognized in England so that certain trades are scheduled as coming under the workmen's compensation act. It is said, however, that addiction to alcohol tends to diminish such personal hygiene as would readily prevent the prolonged action of the materials used. Workers with aniline oil frequently develop carcinoma of the bladder and it is believed that this substance in the process of excretion remains for a time in the bladder and so excites the proliferation which follows.[26] Certain individuals also show a peculiarly marked reaction to arsenic so that those trades in which arsenical compounds are used would prove dangerous in such instances. Osler states that Cobalt miners are said to be prone to primary carcinoma of the lung, an organ which is ordinarily rarely the primary site of cancer.

Medico-legal Aspect.—The medico-legal aspect of the subject is of some importance. Usually no more than a contributory influence is to be claimed with respect to injury in the causation of new growth. In a limited number of trades and occupations, however, the careless use of materials such as certain coal-tar products or physical forces such as the X-ray and radium should always be emphasized and measures taken to avoid all unnecessary exposure. The dangers in the use of X-ray are at the present time so well understood that it would appear that the majority of injuries produced should be considered as due to ignorance or carelessness, whether the injury effects operator or patient. There are as yet no recorded cases of carcinoma arising from radium burns but the similarity of the more penetrating rays of radium and of its biological effects leads to the expectation of similar results and to a warning against the indiscriminate or careless use of this agent.

This book was received while revising the page proof of this Chapter. Dr. Hoffman[27] devotes 28 pages to the mortality from cancer in different occupations and presents an excellent review of the literature of the subject with special reference to cancer among chimney sweeps, gardners, petroleum industry and paraffin workers, coal, soot, tar and pitch workers, including the patent fuel industry, brewers, furriers, skinners, seamen, tinplate workers, lead workers, rubber workers, chemical workers and X-ray workers. The relation of cancer to injuries is emphasized by quotations from Greer's treatise on Industrial Diseases and accidents.

A very valuable reference on the effect of the synthetic dye industry on the occurrence of tumors by Dr. S. G. Leuenberger of Zurich will be found on page 68. Another valuable contribution is referred to on pages 61 and 68.

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It deals with the remarkable prevalence of malignant disease of the lungs in the miners in the Schneeberg district in Saxony. The minerals mined being mostly nickel, cobalt and bismuth. Härting and Hesse in 1878 reported that a lymphosarcoma of the bronchial lymph nodes, or an endothelial sarcoma was responsible for 75 per cent. of all the deaths among the miners. Arnstein in a recent investigation found that between 1907–1911 all the miners admitted to the hospital, entered with a diagnosis of cancer of the lung and it was given as the cause of death in 44 per cent. of the death certificates. In the opinion of Arnstein it is probable that in many cases tuberculosis and possibly also pneumoconiosis may have been erroneously diagnosed as cancer of the lung as necropsies are rare. In the two cases which he was able to examine post-mortem, the trouble proved to be chronic pulmonary tuberculosis in one case but in the other true carcinoma of the lung with metastasis. He urges further study. See also J. A. M. A., June 28, 1913.

The English mortality statistics referred to on page 71, bring out the fact that "the highest standardized cancer death rate occurred among chimney sweeps for which occupation the rate was 224.9 per 100,000 exposed to risk. The rate for seamen was 170.5 and for brewers 166.6. Relatively high, but not abnormally excessive, cancer death rates are met with in the following occupations: Fishermen 111.9; tailors 112.9; textile workers 112.6; lawyers 111.8; innkeepers 108.8; corn-millers 105.3; gas works service 107.1; shoemakers 103.2; and butchers 102.8. Lower cancer death rates but still suggestive of special predisposing conditions are met with in the following occupations: Farmers and graziers 94.8; farm laborers 79.7; gardners and nurserymen 85.2."

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2. X-RAY. RADIUM

X-ray

Exposure to the Röntgen rays, more particularly the repeated exposure of X-ray operators without adequate protection, over a long period of time, has produced skin lesions which have in numerous instances eventually been followed by true cancer. Thus the causal relation of the X-ray and cancer is now a well-known and an established fact.

It has long been known that characteristic burns might be produced by exposure to the X-ray. These burns differ in many respects from those caused by heat; there may be a considerable latent period between the exposure and the subsequent appearance of the inflammatory reaction. This interval may vary from a few hours to several days or even weeks; the burn is of longer duration, the healing may be very slow, requiring weeks and even many months; in some instances healing does not occur, and there may be a persistent chronic ulcer which may become cancerous, particularly during scar formation.

The *degree* of the acute reaction following exposure to the X-rays is very variable, depending on several factors, namely: the intensity and quality of the radiation, the duration of the exposure, the part of the body exposed, and the individual sensitiveness or tolerance. The intensity and quality of the rays depend upon the amperage, voltage, the distance from the tube and the filtration and protection. Gocht[1] claims there is no special idiosyncrasy to the X-ray as was at one time believed. The effects depend on the quantity of rays absorbed by the tissue.

Acute X-ray Dermatitis.—Depending on the factors above referred to, acute X-ray dermatitis may appear as a slight or intense erythema varying in shade from a light pink color, which is more or less transitory, and may have a slightly punctate character, to a deep red erythema, which may not disappear even on pressure. This is often accompanied by itching. The erythema, if intense, may fade slowly and be followed by brownish pigmentation. On the other hand vesiculation may result, with subsequent exfoliation of the surface layers. Ulceration in varying degree and even sloughing may occur. The acute reactions most frequently occur in patients exposed to the X-ray for diagnosis or treatment; such injuries have caused much suffering and disability, and many legal suits, often most disastrous to physicians, have resulted.[2]

The mucous and serous surfaces seem to be more resistant to injury by the X-ray than the skin, while the appendages of the skin are even more

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easily affected. Marked changes in the hair follicles often lead to falling out of the hair; dryness of the skin due to injury of the sweat glands is common.

Normal living tissues are said to be affected in the following order: lymphoid tissue, epithelial cells of the glands, the hair follicles, skin, lining of the blood-vessels, and to a much less extent the cells and fibers of muscles and connective tissue.

From the standpoint of occupational disease, however, the chronic changes caused by repeated exposure to relatively small doses of X-ray over a long period of time are of far more importance than the acute reactions above referred to. They may be discussed as to the local and general effects.

Chronic X-ray Dermatitis and Cancer.—Chronic X-ray dermatitis is the term given to the changes produced in the skin by repeated exposure to

FIG. 21.—Photograph of the left hand and wrist of a Röntgenologist who had been engaged in X-ray work for ten years (Huntington Hospital No. C. O. 14, 240).
The lesions chiefly on the palmar surface, which is unusual, consist of scaling, fissures, keratoses, atrophy, telangiectases and pigmentation.

relatively small doses of X-ray. In certain instances they may be preceded by acute reactions; the characteristic changes are closely analogous to those found in the skin of the aged, or of those exposed to the elements. Similar changes may be produced in the skin of the paraffine worker and the chimney sweep. The atrophy, keratosis and localized scaling is so like that of age, that the chronic effects of the X-ray upon the skin have been claimed by some[8] to be prematurely induced senescence.

In addition to the atrophy, scaling and keratosis, there is also pigmentation, development of fissures and telangiectasis, which may be marked (Fig. 21); loss of hair and dryness of the skin are frequent; then ails may become brittle, ribbed and chipped (Fig. 23). Beneath the keratoses changes occur in the underlying blood-vessels and connective tissue, the epidermis may invade the corium and true carcinoma may result, with metastasis,

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not only in the regional glands, but even widely disseminated. This represents the first real experimental cancer.[4]

Cancer may develop not only in preëxisting papillomata and keratoses, but also in the intractable chronic ulcers caused by the X-ray, either by intense single or repeated exposures. Many cases are reported of the development of cancer in patients who have undergone X-ray treatment for

FIG. 22.—Photograph of an X-ray demonstrator showing on the chest keratoses, telangiectases and pigmentation; on the hands keratoses, fissures and loss of left fore-finger due to previous amputation for X-ray carcinoma. In the left axillary region a black line indicates the anterior border of a metastatic cancer mass. These chest lesions were caused by repeatedly demonstrating the heart outline and action by fluoroscope and the lesions on the left hand were due to the use of the latter for a penetrometer and as an object of demonstration for bone structure. Man first began X-ray work in 1897. The case was previously reported by Porter, No. VII. of his 1909 series, also Huntington Hospital, No. C. O. 15. 1. Jan., 1915.

lupus; indeed, this disease may in certain instances be followed by cancer, even without treatment by the X-ray. Mendes da Costa[5] cites 71 cases of lupus in which the X-ray was used; of these seven became cancerous. Schurmann[6] thinks the danger of cancer from X-ray treatment of lupus is slight when compared with its extensive use and the fact that cancer may occur in lupus without the use of the X-ray.

The pioneer workers with the X-ray little suspected the dangers of occu-

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pational disease and the late occurrence of serious injury among them is appalling (Figs. 22, 23, 24). Many cases have now been reported which emphasize the importance of adequate protection and care in working with the X-ray. Porter[7] in a most admirable article has reported a considerable number of cases, and discussed methods of treatment. Of the symptoms caused by X-ray injuries *pain* is most distressing; in certain instances it is agonizing; disability may be great.

Goeneu[8] found a mortality of 24 per cent. in 33 cases of X-ray carcinomata gathered from the literature, while of ten cases reported by Porter and White[9]

FIG. 23.—Photograph showing details of Fig. 22, *i.e.*, telangiectases, keratoses and pigmentation of chest, the bulbous stump of the left fore-finger, the multiple keratoses, crusts, and fissures, and the chipping and ribbing of the nails on the left hand, which is laid across the chest.

six died in spite of surgical operation. Various other writers have reported single or a series of cases of X-ray carcinoma.[10]

It is important to note that these X-ray carcinomata are frequently multiple,[11] there are usually several centers of growth.[16]

Sarcoma[11] as well as carcinoma may result from exposure to the X-rays.

Of the 36 cases of X-ray carcinomata collected by Porter,[7] 34 were in professional X-ray workers (Figs. 22, 23, 24), and two in patients, the latter with abdominal tumors which had been treated for a long time.

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Histology of Chronic X-ray Dermatitis and Carcinoma.—Wyss,[4] Unna,[3] and Wolbach,[12] agree that the epithelium is not primarily affected but that the essential changes are in the underlying connective tissue and blood-vessels. There is sufficient injury so that complete repair does not take place—degenerative and reparative process go on in the corium but the vascular lesions are progressive. Young fibroblasts were described by Wolbach in the media of the small blood-vessels which, from the proliferation of the connective tissue and the swelling of the collagenous material, may be in certain instances obliterated.

FIG. 24.—Photograph showing ulcerations on chest of a demonstrator of X-ray apparatus due to the removal of numerous X-ray keratoses by actual cautery; between these pigmentation and telangiectases are seen. The stump of the fore-finger the site of a previous X-ray cancer, and the middle finger have been amputated because of recurrence; the scar in the left axilla indicates where a large, metastatic cancer mass was removed; the primary growth was on the left fore-finger. Feb. 8, 1915.

The necrotic foci found in both the superficial and deep corium are probably of vascular origin. The cells in the basal layers of the epithelium may show many mitoses. Only by the downgrowth of the epithelium is it able to reach viable connective tissue. The glands are atrophied and the fat tissue is absorbed and replaced by connective tissue.

Hyperkeratosis, rarification of the corium, irregularity of the papillæ are common; cell infiltration, especially lymphoid and plasma, is variable. Epithelium may be found growing into necrotic foci and into thrombosed

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telangiectases; in more advanced cases epithelial pearls situated in the deeper structures are frequent, giving an appearance differing in no way, histologically, from a true epidermoid carcinoma.

General Effects of the X-ray.—Much less is known of the deeper and more general chronic effects due to exposure to the X-ray. It has, however, been definitely established that the sexual glands are particularly affected. Atrophy of the ovaries and testicles may occur with resulting sterility; changes take place also in the blood, lymphatic tissue and bone marrow. Anæmia, even of the pernicious type, may be produced and blood findings, similar to those described from exposure to radium, are frequent, particularly in the form of a relative lymphocytosis.[13] On the other hand, it has been claimed that certain cases of leukemia have resulted from exposure to X-ray.

In a series of mice and rats exposed repeatedly to the X-rays, marked changes were found in the lymphatic tissue; the spleen was markedly affected being very much reduced in size due to destruction of the Malpighian corpuscles and other cellular elements.[14] In such animals resistance to infection is markedly decreased.[15] Certain cases of myelogenous leukemia, after exposure of the spleen to the X-ray, if special precaution is taken regarding screening, protection and dosage show a marked reduction in the size of the greatly enlarged spleen, great decrease in the white-cell count, and more or less temporary improvement in the general condition of the patient. Leucocytolytic substances are said to have been found in the blood of such cases.

Toxæmia of varying degree may be produced by exposure to the Röntgen ray, particularly in patients in whom there is poor elimination by the kidneys. Slight toxæmias have been noted by Edsall[16] even after diagnostic exposures in such patients. Profound, even fatal, toxæmias have been recorded; such a severe case with resulting stupor has been personally observed by us.

This toxæmia may be the result of poisoning from failure to eliminate products of protein metabolism. There may be increase of total nitrogen, ammonia, urea in the urine, and nitrogen retention in the blood. On purin free diet there is to be found an increase in endogenous protein metabolism.

Other general effects of the X-ray are said to occur; lassitude is of common occurrence, frequently patients doze or even fall asleep while undergoing X-ray treatment and there is often a feeling of undue fatigue after long exposure. Alopecia is common in X-ray operators and patients exposed.

The details of the pathological findings have been described by Cavazzeni and Minelli[17] in a post-mortem examination of one who had been exposed to the X-ray for many years.

Treatment.—The best treatment for acute Röntgen dermatitis is said by Dodd[18] to be the liberal application of whitewash which is sopped on the inflamed area but not to be used with a dressing. He decries the use of ointments. The formula given by Dodd is as follows:

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℞.	Zinc oxide	30	℥i
	Phenol	2	ʒss
	Glycerine	4	ʒi
	Limewater	240	℥viii

Sig.—Shake well and bathe area for 5 to 10 minutes, twice or three times a day.

For slight burns Unna's zinc gelatine, and for the more chronic conditions on the hands compound tincture of benzoin, may be used.

The chronic changes produced in the skin by the X-ray require varied treatment depending on their nature. The so-called precancerous keratoses, papillomata and ulcers, call for excision, curettage, or skin-grafting, and the more border-line and cancerous lesions may demand amputation. Porter[7] in the excellent article above referred to describes the treatment of these lesions in detail. Recently Caldwell[19] has called attention to the value of surface applications of radium in the less severe and strictly cutaneous chronic lesions, such as fissures, persistent scaling and keratoses. Freezing methods and the electric spark, are more painful.

Of even greater importance than treatment in individual cases is the matter of preventing these injurious effects of the X-ray.

Prophylaxis.—As our knowledge of these injurious effects has increased, many methods have been devised to prevent exposing the body of the operator to the rays, and of adequately protecting the patient. For the X-ray worker mirrors, cords and pulleys have been planned for indirect observation of the patients and control of apparatus. Protection for the operator in the form of leaded cabinets, screens, gloves, aprons, etc., have been provided. These are well illustrated in special works in Röntgenology and in catalogues of dealers in X-ray apparatus.

The patient is now more carefully guarded by screens, leaded cloth and more particularly by our knowledge of dosage, filtration and protection, each varying with the object of the treatment. Exposures should be as short as possible and other forms of irritation of the skin avoided. The details of the appropriate technique will be found in the general or special works on Röntgenology and in its application to the diagnosis and treatment of pathological conditions of the various special organs for which such procedure is of value.

That such prophylactic measures are adequate is probable from the fact that those who have carefully used such measures though engaged in X-ray work for over 12 years have not suffered any harmful effect.

RADIUM

The increasing use of large quantities of radium for therapeutic purposes makes it important to describe in detail the symptoms and signs produced by it and to emphasize the importance of these as occupational injuries so that more serious late effects may not result.

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Rutherford[20] states that "Walkhoff first observed that radium rays produce burns of much the same character as those caused by Röntgen rays. Experiments in this direction have been made by Giesel, Curie and Becquerel and others with very similar results. There is at first a painful irritation, then inflammation sets in, which lasts from 10 to 20 days. This effect is produced by all preparations of radium, and appears to be due mainly to the α and β rays. Care has to be taken in handling radium on account of the painful inflammation set up by the rays. If a finger is held for some minutes at the base of a capsule containing a radium preparation, the skin becomes inflamed for about 15 days and then peels off. The painful feeling does not disappear for 2 months."

Although these rather *acute* reactions due to radium are apparently well known to *physicists*, working with radioactive substances, and considerable experimental work has been done on animals by biologists, very little attention has been paid to the more chronic changes.

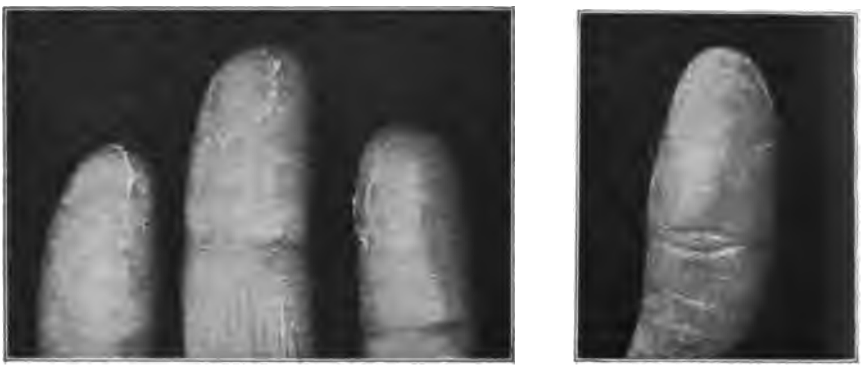

FIG. 25.—Photograph of the thumb, index-, middle- and ring-fingers of the left hand showing the scaling of the thickened superficial layer of the skin on the radial side of the terminal phalanges; this is due to the handling of radium in making therapeutic applications.

The increasing use of large quantities of radium should call attention to these changes not only because of the annoyance and discomfort caused but as a warning from analogy to the X-ray of more serious late effects, such as atrophy, intractable ulceration and even cancer. Already in certain instances there has been caused not only great annoyance from discomfort but actual impairment in manual dexterity in performing delicate manipulations because of persistent local anæsthetic effects.

The symptoms caused by handling radium may occur very insidiously and consist of blunting of sensibility of the finger tips, paræsthesia such as increased sensitiveness to heat and pressure, amounting at times to actual pain, and anæsthesia of varying degree.

The subjective disturbances are out of all proportion to the objective findings which include flattening of the natural ridges on the effected fingers

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with consequent changes in the characteristic markings of the finger prints, thickening of the horny layer of the epidermis with scaling in varying degree (Fig. 25), failure of the tips of the fingers to resume their normal shape after pressure—a sort of pitting, upgrowth of the cuticle at the base of and underneath the nails which tend to stand off from the fleshy part of the fingers and which become easily cracked and extremely brittle (Fig. 26).

Various general symptoms such as headache, malaise, weakness, undue fatigue, unusual need of sleep, increased excitability, fretfulness, irritability, disorders of menstruation, attacks of dizziness, etc., have been said by Gudzent and Halberstædter[21] to be caused by repeated and long-continued exposure to radioactive substances. Such symptoms are, however, common

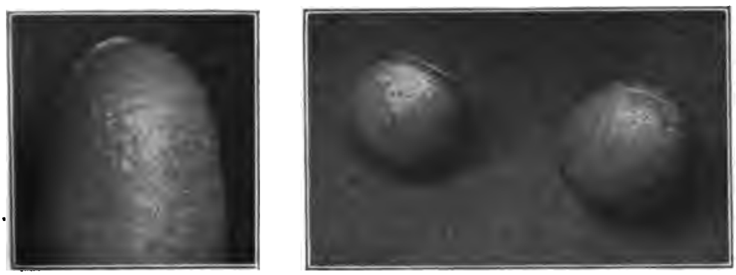

FIG. 26.—Photographs of fingers showing how the free edge of the nails stands away from the fleshy part of the affected finger tips; the upgrowth of the horny skin beneath the nails is marked. The changes are the result of handling radium in making therapeutic applications.

in many people at times and as they cannot be accurately and objectively recorded there is doubt if they can be definitely proved to be due to exposure to radium. They may be due to close confinement, tiring routine and lack of out-door exercise and other causes. The exposures of some of the cases reported were doubtless large; some were assistants in "Fabriks" for manufacture of radium apparatus and some had been engaged for years during the entire day in work with radioactive substances. It is therefore probable that certain general symptoms do occur as a result of exposure.

Changes in the blood of radium workers were observed by Gudzent and Halberstædter. Most striking was the relative and absolute increase in lymphocytes from 36 per cent. to 63 per cent., average of 10 cases 46.4 per cent.; a relative and absolute decrease in neutrophiles, average 50.3 per cent. There was little change in red blood corpuscles, slight diminution in white cells and the hemoglobin was lowered in only two cases, 70 per cent. and 71 per cent. respectively.

Various methods have been devised for avoiding these injurious effects by the least possible contact of the fingers with the radium. Forceps or special vices are used for holding tubes and screwing in or out the tips and eyes; special applicators in the form of metal boxes have been constructed

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so that the active tubes may be added after the filtration and protection have been arranged, and the surface applicator is then slipped by forceps into a special rubber envelope and fastened with adhesive. This is particularly to avoid wrapping the radium up by hand in sheet rubber. Leaded gloves, fingers, etc., are clumsy and are not readily worn.

In placing active tubes in special applicators it will probably not be possible to avoid all contact with radium and as the effects are not apparent at once, as when handling very hot objects such as heated glass, but only after a period of days or even weeks, it will be difficult to train a worker to avoid all contact with the active apparatus.

In the work of making routine applications of radium there should be a rotation in the staff and persons effected should be freed at least temporarily from such work.

In order to avoid general disturbances the body should be protected as far as possible by metal screens in form of boxes or plates about the radium; there should be frequent ventilation of workrooms, particularly if there is radium emanation present, and a change of duty and shorter hours; periodic physical examination of those working with radioactive substances with special reference to the blood examination is indictaed.

Summary and Conclusions.—From the above it is evident that marked changes may occur on the fingers of those engaged in routine work with radioactive substances. These local objective changes consist chiefly of flattening of the characteristic ridges, thickening and scaling of the superficial layers of the skin and even atrophy and intractible ulceration. These lesions are usually slight compared with the marked subjective symptoms, such as paræsthesia, anæsthesia of varying degree, tenderness, throbbing and even pain. The persistence of such effects is noteworthy.

Various general systemic symptoms and also blood changes may be produced by exposure to radioactive substances. To avoid such local and general disturbances special protective and preventive measures have been devised and those engaged in routine handling of radioactive substances are particularly cautioned.

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DIVISION VI

Electrical Injuries and Electrical Shock

BY SIR THOMAS OLIVER, Newcastle-upon-Tyne, England

Man's equipment has in recent years been considerably added to by his mastery over electricity. By utilizing the potent fluid, he has achieved results which have transcended the imagination of his forebears of a century ago. Distance has been abridged and the atmosphere caused inaudibly to vibrate with the winged messages of human thought. While electricity has aided civilization and been of the greatest assistance to man, it has brought not only light and comfort into our homes but dangers as well. Electricity is a subtle fluid; we can store it and control its flow, although we know little or nothing about it. In this article we are concerned solely with the medical aspects of the subject. In Europe during 1910 there were 200 deaths caused by electrical shock. Of these 41 were in France and 26 in Great Britain. In America the number was probably larger. In Great Britain according to G. Scott Ram,[1] H. M. Electrical Inspector of Factories, the electrical accidents for 1913 in electrical generating stations alone were 65 with two deaths. Of these the largest number 31, with one death, occurred during the cleaning and repairing of "live" switchboards and "live" conductors. Twelve accidents occurred at switchboards during routine work. Of the 47.4 per cent. of the accidents in 1913 occurring during cleaning and repairing, many of them, with care, could have been prevented. Considering the number of electrical stations in Great Britain and the increasing demands upon their production the number of electrical accidents in the United Kingdom is not considered to be unduly high. To the accidents already recorded there occurred other 418 electrical accidents with 17 deaths in factories other than generating stations. Five deaths were due in 1913 to conductors having no protection at all. Trimming of arc lights was the cause of three accidents with one death. Of 20 fatalities reported to the Home Office for 1913 as coming under the Factory and Workshops Act, 15 were due to shock, and 12 of these 15 were due to alternating currents at pressures which are usually considered low. Two of the deaths were the result of burns. In one of the patients blood poisoning followed external injuries.

The average age of 63 out of 66 persons injured in electrical generating stations was just over 20 years, a circumstance which shows that important

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operations such as testing should not be handed over to apprentices or to pupils who have little experience of the danger which is incurred. Many of the accidents are no doubt unavoidable, but many can be prevented. Some of the common causes of accidents are mistakes in switching, failure to disconnect and to earth test leads before handling them, also cheap installation. Insulated spanners should be provided by employers, also India-rubber gloves. The absence of insulated spanners was during 1913 in Great Britain the cause of 13 non-fatal accidents. The nature of the floor, too, in which electricity is generated is of importance. In one instance an installation plant was found in a building with an iron floor.

Death in electric shock may be instantaneous. Three theories at least have been advanced to explain the fatal event: (1) inhibition of the respiratory center whereby breathing ceases (D'Arsonval) or of the cardiac center whereby the heart stops beating (Bourrot); (2) sudden and complete cardiac failure (Tatum, Prevost-Batelli); (3) hemorrhages or acute disruption of nerve cells (Donlin, Icllinck).

Dealing with those cases where death does not supervene until several minutes or a few hours after the accident, Prof. J. P. Langlois[2] expresses the opinion, not necessarily his own, that the death is probably to be explained by bulbar and bulbo-spinal paralysis due to structural alterations of the nerve cells, or by air embolism consequent upon chemical changes in the tissues induced directly by the electrical current or indirectly through changes in the trophic action of nerves.

Before concluding under what circumstances an electrical current is likely to prove dangerous to an individual brought into contact with it, there are a few things which have to be thought of. There is the current itself, its pressure and intensity, its nature, *e.g.*, alternating or continuous, the duration of the contact, resistance of the conductor, the position of the electrodes on the body and the insulated position of the individual at the time he received the shock.

By the term voltage we mean pressure or tension and by amperage intensity. It is difficult to say what voltage is fatal to man. Under some circumstances 240 volts may prove fatal, under other circumstances 1000 volts may do no harm. Much depends upon the conditions under which the current is received. Of the two kinds of electric current, "continuous" and "alternating," the "alternating" is the more dangerous. In Great Britain during 1902–1908 for three deaths caused by continuous currents 30 were due to alternating. Circumstances such as these, however, do not absolutely prove that alternating currents are the more dangerous. On the other hand, the British Board of Trade considers alternating currents to be twice as dangerous as continuous, while Weiss regards alternating currents as four times more dangerous than continuous. The duration of the contact is of importance. Prevost and Batelli state that the continuous current requires less time than the alternating: one-tenth of a second is sufficient for the con-

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tinuous but the alternating requires three-tenths of a second. When a man comes into contact with live metal one or more than one of three things happens: (1) he is burnt, (2) rendered unconscious and is convulsed or (3) he is killed instantaneously. Persons apparently killed by electric shock may be restored to life by artificial respiration. An electrocuted person is an asphyxiated person.

The consequences of the passage of an electrical current through the body are proportional to the quantity of current passed and the conditions under which contact is made. A moist skin intensifies the action of a current. A dry skin, on the other hand, may prevent serious consequences. Trotter found when wearing a pair of dry boots a continuous current of 500 volts produced hardly any effects. He took a walk through the wet streets of the town and found that with the same current, 500 volts, the milliamperage had risen and that the resistance of the boots had fallen 15 per cent. Given fairly good contact a low voltage may be sufficient to throw the muscles of the body into such a state of tetanic rigidity that it is impossible for the individual to breathe and to relax his grasp of any charged metal he may have taken hold of. To release such a person the circuit must be broken. Since it is when currents enter and leave the body that effects take place, the "making" and "breaking" of contact are the dangerous occasions. Severe pain may be the result of the entrance of an electrical current owing to the strong muscular spasm induced, the electrolytic action upon nerves or of the action of the products of electrolytic decomposition. Should the individual not be immediately thrown into a state of unconsciousness a condition of terror is produced which may not only cause the victim to swoon but the mental effect may be such as to cause him to be nervous and apprehensive for months afterward.

Burning is one of the visible consequences of contact with electric currents of high potentiality. The skin is charred; if open wounds have been caused they are irregular and deep; the bone is frequently exposed; the sores are angry looking and are disposed to slough. They are painful and are slow to heal, probably owing to destruction of terminal nerve fibers. Burns occur when the action of the current has been local. A current sufficient to cause severe burns might, if it traversed the body, cause death. There are cases where, after contact with high electrical currents, little or no effect is seen on the skin but internal damage has been inflicted upon the deep tissues. In a youth in whom this had taken place there were no external signs of injury, but as a consequence of deeply seated effects followed by gangrene the limb had to be amputated below the knee. Later, owing to sloughing of the deeply seated tissues of the thigh, the remaining portion of the leg had to be amputated. A definite opinion therefore as to the extent of the injuries cannot always be given on a primary and superficial examination. One man whom I saw in consultation had, while crossing an electrified railway, inadvertently touched a live rail with his wet boot. He was immediately thrown down. Fortu-

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nately the accident occurred close to a signal box and was witnessed by the man on duty. Leaving the box and putting on India rubber gloves the signal man succeeded in removing the injured person from the rails. Patient was unconscious. Help was secured and an ambulance obtained. On the road home the injured man while still insensible had a series of convulsions. Upon a cursory examination no external injuries were observed. It was not until his home was reached and his boots, which were uninjured, removed that the toes and front of each foot were found to be severely burned. In an hour or two consciousness returned. For several days the feet were extremely painful. The wounds were deep and angry looking; the bones of the big toes and of the metatarsus were exposed. Healing was slow. I also saw a youth aged 19, the subject of severe burns, who received a shock of 6000 volts, alternating current, without fatal consequences. His right hand was almost burned through. The hand was charred, irritable looking and disposed to slough. The outer three fingers were destroyed. In addition there were acutely inflamed and blistered patches on the lower part of the right forearm, also blisters on the soles of the feet. Patient's boots were uninjured. This youth was working on the three-phase system; he had touched one wire and then another without any consequences, but on touching the third wire he fell and became unconscious. He was standing on a concrete floor at the time.

One of the worst cases I have seen was that of a young laborer admitted into the Royal Victoria Infirmary, Newcastle, under the care of my surgical colleague, Professor H. B. Angus. The man had accidentally made contact with a current of 20,000 volts. I saw patient 2 hours after the accident. He was unconscious but kept rolling about in bed in an extremely restless manner. His pupils were widely dilated. There were several large burns on the trunk, front and back, also on the limbs. There was a large circular burn on the back of the head with exposure of the occipital bone. He died 7 hours after receiving the shock. At the autopsy, 40 hours after death, there were hemorrhages in the occipital lobe of the brain, numerous minute hemorrhages on the visceral layer of the pericardium; liver, kidneys and portions of the lungs were deeply congested.

In both of these cases the voltage was high. As an illustration of a comparatively low pressure—110 volts—causing death, the following note, taken from the pen of Dr. Fleury[3] of Versailles, may be quoted. It is the case of a healthy married woman aged 35 who died in 25 seconds after receiving the shock. She was in the act of having her usual daily bath. The bath, which was made of enamelled iron, was closed by a metallic plug placed in the middle of the floor of the bath. The plug was attached to a chain and to this a float. An electric bell communicator, composed of a chain of metallic rings ending in a porcelain handle, was fixed to the wall close to the bath. Madame C after having been attended to by her maid was left to complete her bath. Hardly had the maid left when hearing a cry she rushed back to the bathroom where she found her mistress with her head out of the water but fallen forward,

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the face cyanosed and both hands spasmodically grasping the chain of the electric bell. In attempting to remove the chain the maid herself received a severe electric shock. Madam C was dead. All efforts to restore her proved unavailing. The whole tragedy was enacted within 25 seconds. It is presumed that Madam C must have, when in the bath, formed an electric circuit between the left hip which was resting on the metal plug in the bath and the chain of the electric bell which she was grasping in her hand at the time of her death. The current when tested was found to be capable of lighting a lamp of 110 volts. On examining the body after death two small hemorrhages were observed on the upper and posterior part of both arms near the axillæ, and on the left hip there were a few blisters filled with blood. At the postmortem examination nothing special was found in the heart, pericardium, the abdomen and brain, while beyond a few limited areas of congestion and a few old pleuritic adhesions the lungs presented nothing abnormal. The appearances generally speaking, but especially those presented by the heart, were those of asphyxia. The left ventricle was empty, the right ventricle was filled with dark non-coagulated blood. The low voltage, 110, was at first thought to be an unlikely cause of death, and yet everything pointed to death from electric shock.

As already stated, pressure or tension, that is the number of volts, is one of the factors to be considered in electrical injuries. Prevost and Batelli of Geneva are of the opinion that voltage below 100 is only slightly dangerous, and yet death has followed contact with a current of 75 volts. The intensity of an electrical current is represented by and for the purpose of illustration is regulated much in the same way as the flow of water when a tap is turned on. The intensity is calculated in amperes. From a medical point of view what is important is the intensity of the current as it traverses the body at the time of the accident. A current of 25 milliamperes is bearable; one between 25 and 60 is capable of doing considerable harm; beyond this the danger increases proportionally, death almost always supervening if the intensity reaches 100 milliamperes.

The resistance offered by the body to the passage of the current has also to be considered. A relationship is thus established between the three factors: tension (E), intensity (I), and resistance (R). This, known as Ohm's law, is expressed thus:

$$I = \frac{E}{R}$$

by which is meant that the intensity in amperes is proportional to the tension in volts and is inversely proportional to the resistance in ohms. In other words, the feebler the resistance offered by the subject the greater will be the intensity in amperage and the more likely will the subject be affected by the electrical current. Such other factors as already mentioned, viz., the extent of the surface of the body exposed to the current and the

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dryness or humidity of the skin, cannot be ignored. A similar remark applies to the period of time during which the current passes. Resistance becomes diminished as the current traverses a body and as a consequence the intensity in amperage rises. In illustration of this, Fleury in his experiments found that while a dog could resist a current of 4000 volts during 2 seconds, it succumbed to contact with 110 volts applied for 19 seconds.

The direction of the current, too, is of importance. Should the heart lie in the path of the current, danger to life is enormously increased; hence the risk of a current when it is passed through hands to feet and *vice versa*. The following cases recorded by Fleury are two of the fatalities which have been caused by what might be considered low voltages. A youth received a shock of 125 volts; he fell to the ground and in rolling over caused the electric wire to encircle a metal bucket which was being carried by a woman standing close by. The woman was killed instantaneously. A female cook in Geneva attempted while one of her wet hands held an electric lamp to draw water from a lead pipe with the other. She had hardly touched the tap when she received a shock from a current of 110 volts and succumbed almost immediately. Since death has been caused by voltages of 110 to 170 in the case of persons completing circuit through touching electric lamps in dwelling houses, people ought to be forewarned of the dangers.

CAUSE OF DEATH IN ELECTRIC SHOCK

In order to ascertain the cause of death in electric shock I carried out a few years ago a series of experiments with my colleague, Prof. R. A. Bolam, upon anæsthetized dogs and rabbits, the continuous current being employed. Two of the main theories of the cause of death are: (1) arrest of respiration; (2) stoppage of the heart. In order to ascertain which of these two is the cause of death, we had arranged before bringing the animals into contact with the electrical current that a record of the blood pressure and respiratory movements should be taken simultaneously. In this way we sought to demonstrate how the heart and respiration became affected when high electrical currents are passed through the body. On making electrical contact the animal would at once be thrown into an attitude of opisthotonos, the muscles generally throughout the body becoming rigid. The lever of the apparatus recording the respiratory movements would be suddenly and violently raised, while the other, indicating the blood pressure and beat of the heart, would also be suddenly lifted owing to general arterial constriction. Momentarily maintained in these positions the levers would gradually fall so that their oscillations covered a shorter range. On breaking contact, the respiration would become deeper and quicker than it was before the shock. Thereafter, within a few seconds, breathing and the heart's beat would become normal. In dogs which succumbed rapidly to electrical shock there was the same initial respiratory spasm with a sudden rise of arterial pressure

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followed by an immediate fall. A few further oscillations of the lever marking the arterial pressure would be observed, and then a sudden fall of the lever would occur indicating that the heart had ceased beating. In some of the electrocuted animals respiration continued for several seconds after the heart had stopped. In other experiments with 240 volts, continuous current, death in electrical shock was of cardiac origin and not respiratory. Where an electrical current was not sufficient to kill a dog immediately, the heart's beat would be momentarily delayed, then quickened, during which the cardiac sounds could be heard quite well, but when currents of higher potential were employed the sounds of the heart on contact being made would cease almost immediately while the respiration continued. If artificial respiration was not adopted the breathing gradually ceased and the beat of the heart did not return, the pupils dilated, mucus flowed from the mouth and increasing pallor stole over the body.

On exposing the heart of anæsthetized dogs and inserting a canula into the trachea in order to carry on artificial respiration, we had ocular demonstration that it was the heart which was primarily arrested as a consequence of the passage through the body of continuous electrical currents of 240 volts.

Professor A. M. Bleile of Ohio State University, as the result of experiments, concluded that death in electrical shock is due to the current producing through the nervous system constriction of the arteries whereby there is created a mechanical impediment to the flow of blood which the heart is unable to overcome. Bleile found that when drugs were given beforehand to counteract the contraction of the arteries higher voltages could be borne. Bolam and I came to a similar conclusion. When we caused anæsthetized animals to inhale amyl nitrite before making the electrical contact we noticed that stronger currents could be borne. In our experiments we found that the heart was the organ primarily affected even when atropine had been previously administered to abolish the influence of the vagus. Whether the heart muscle is paralyzed by the electrical current, destroyed by the molecular changes produced by the current, or brought to a standstill by the enormous resistance of the constricted arteries above referred to I am not in a position to say. Not only is there no definite relation between the size and weight of an animal and the strength of the current, but a current which is passed for a few seconds one day without bad effect may on another kill the animal in 2 or 3 seconds.

Fleury found in dogs, when the milliamperage rose to 70–80, that within 5 seconds the heart showed fibrillary tremor. When the current was cut off there was marked fibrillation of the ventricles of the heart and respiration which had ceased recommenced and shortly afterward gradually died away. To explain death in electric shock Dr. G. Weiss advances the following theory: Under the influence of alternating currents the muscles of the body are thrown into a state of tetanus, organic combustion is enormously increased, there is an imperative necessity on the part of the animal to absorb oxygen and to

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exhale carbon dioxide. Respiration is embarrassed owing to tetanic rigidity of the thorax and as a consequence asphyxia develops shortly. Fleury, basing his opinion upon the fatal case alluded to in the text, regards the mechanism of death in electric shock as complex; there are asphyxia and paralysis of the heart. When these occur death is rapid. When death is not instantaneous he is of the opinion that death is brought about by fibrillary tremor of the heart.

J. L. Prevost and F. Battelli of Geneva found with alternating currents of low tension that the heart was thrown into a condition of fibrillary tremor. With a tension of 20–40 volts the heart became paralyzed but respiration continued. When high alternating currents of 240–600 volts were employed the heart and respiration ceased simultaneously. Prevost and Battelli found in using continuous currents that the results were practically the same as with alternating, there being, however, this important difference, that when continuous currents were employed voltages less than 50 did not produce fibrillary arrest of the heart's action, but alternating currents of only 10 volts induced fibrillary tremor followed by stoppage of the beat of the heart.

There is a general consensus of opinion that electrical currents below 150 volts if they prove fatal cause death by stopping the beat of the heart which had been previously thrown into a condition of fibrillary tremor. High tension currents, *e.g.*, 1200 volts, leave the heart intact, but they arrest breathing and induce asphyxia which if not treated may end in death. With voltages between these two, sometimes the heart ceases beating, sometimes respiration is arrested or the two events occur simultaneously. Experience shows that such low voltages as 75–100 are not free from danger.

PATHOLOGY

If the body is examined shortly after death there is usually rigidity of the muscles. There may or may not be external signs of burning. The body may be pale or livid. On opening the body cavities the abdominal veins are found filled with dark liquid blood and the viscera are observed to be congested. The right ventricle of the heart is flaccid and the left ventricle hard and tense. In the right side of the heart the blood is dark and liquid; the left ventricle is practically empty but the left auricle usually contains dark liquid blood. The absence of coagulation of the blood is noteworthy. This is of great significance, but it is not absolutely diagnostic as in some of my experiments I found coagula in the right side of the heart. Beyond a few ecchymoses and signs of congestion the lungs may show no special feature. Generally speaking, the signs are those of asphyxia. The brain and spinal cord may be congested or they may show no abnormality. The blood on spectroscopic examination was found to contain in some of my cases both oxyhemoglobin and reduced hemoglobin. Usually the pupils are dilated. On examining the brain of one of the patients killed by currents of high poten-

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tiality there were observed minute hemorrhages due to rupture of small vessels in the cortex and white matter. The blood showed evidences of hemolysis. Drs. F. W. Mott and Schuster, who were kind enough to examine sections of the brain microscopically for me, found chromolytic changes in the brain cells. Similar changes were noticed in the cells of the medulla oblongata.

As bearing upon the lesions found in the brain, mention must be made of the important contribution to this subject by Drs. E. A. Spitzka[4] and H. E. Raschi of Jefferson Medical College, Philadelphia. Their findings are of importance since the lesions described obtained in the brain and spinal cord of five criminals who had been electrocuted—the voltages varying from 1750 to 1850, amperage 8 to 11 and the contacts two to four. The brain in each instance was removed 15 minutes after death so that there was no time for post-mortem decomposition to have set in. The parts of the brain examined were the mid-brain and the pons oblongata. At the levels mentioned Spitzka and Raschi found peculiar areas, circular in outline and varying in size from 25 to 300 μ. They consisted of two portions—a central rarified zone and a peripheral condensed zone. The areas contained a blood-vessel surrounded by a delicate reticulum, the fibrils of which were radically directed. Nuclei were observed along their course. The peripheral zone immediately in contact with the central stained more readily and deeply than the external. In the peripheral zone there was an absence of nuclei. In the upper part of the spinal cord close to the pyramidal decussation the myelin did not stain well with hematoxylin and eosin, Van Gieson or Weigert's staining fluid. Along the course of the blood-vessels were bead-like expansions like clear beads adherent to the wall of the vessel. The writers are of the opinion that the bead-like expansions, the condensation zone limiting these, the radically disposed fibers and the torn tissues indicate a sudden liberation of gas bubbles due to the electrolytic properties of the current seeking paths of least resistance along the course of the blood-vessels.

Treatment.—Where the electrical shock has been slight and the individual has not been rendered unconscious no treatment is called for. The effects pass away almost immediately. Some persons thus injured remain nervous for several days or weeks afterward. Burns or wounds caused by electricity must be treated on ordinary surgical lines. Where the heart has ceased beating and respiration has been arrested the treatment which is most likely to give satisfactory results is artificial respiration. It matters little so far as this matter of treatment is concerned whether death is primarily of cardiac or respiratory origin. Artificial respiration must be carried on for a considerable time. In the case of a man who had accidentally been electrocuted and who was seen shortly afterward by a medical man, artificial respiration was under his supervision carried on for at least half an hour without any response. Believing that apparent death had been actual the medical man pronounced life to be extinct, and left the place. The workingmen in the factory of

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their own accord carried on artificial respiration for a considerable time after the doctor left and were rewarded about an hour after the accident by seeing their comrade restored to life. Twice Bolam and I succeeded in resuscitating a dog whose heart had ceased beating once for 13 minutes and on the second occasion for 8. The heart was exposed to view so that we could follow the events which took place. The heart had become rapidly distended with blood so as to bulge out the pericardium and had become perfectly motionless after having passed through a stage of fibrillary tremor. By persisting in artificial respiration, aided subsequently by occasional spontaneous inspirations, also through rhythmic traction of the tongue, the contents of the right side of the heart were gradually aspirated into and through the lungs and the auricular beats were reëstablished, at first irregularly and feebly. By degrees the auricular pulsations became stronger and passed over into the ventricles so that after 13 minutes, during which the left ventricle had remained irresponsive, Bolam and I had the satisfaction of seeing the normal beat of the heart restored, the pulmonary and general circulation reëstablished and life return. Leduc points out that when an animal has been apparently killed by electricity and the heart has ceased beating, the animal can usually be restored to life by applying the same amount of current to the body, for since this produces immediate contraction of the muscles of the body, including those of inspiration, artificial respiration is to some extent aided and can be proceeded with. There is a belief on the part of some physiologists, where death is of cardiac origin and has been preceded by fibrillary contraction, that neither artificial respiration nor massage of the heart is of the slightest value in restoring the victim. Artificial respiration upon one of our animals in whom fibrillary tremor had occurred was, as already indicated, successful, but setting that aside for the moment, yet still insisting that artificial respiration is a mode of treatment well worthy of adoption, the following may be not less successful. It has been demonstrated that where a dog has been killed by electric currents of high potentiality and the heart still shows signs of fibrillary tremor, the application directly to the heart of an electrode carrying a still larger current than the one which apparently proved fatal will cause cessation of the fibrillary movements and on withdrawal of the current the heart will resume its rhythmic beats again. In some cases the sufferer is killed outright so that all attempts at resuscitation fail. Under all circumstances, however, where persons are the subjects of apparent death from electrical shock artificial respiration should be resorted to and continued for at least an hour. Workmen in their efforts to rescue a comrade should be provided with India-rubber gloves and insulated spanners, or where these are unattainable should wrap thick, coarse, dry clothing round their hands, for otherwise in their attempts to rescue a stricken fellow-workman they themselves may receive a fatal shock. To cut a live wire long shears with wooden handles should be used.

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ELECTRICA OPHTHALMIA

Electricity is used in the welding of metals. In this operation the brightness of the glare causes injury to the eyesight. Dr. George L. Apfelbach[5] draws attention to a morbid eye condition, *electrica ophthalmia*, caused by electric light rays. In these rays the ultra-violet predominate. The signs and symptoms of the malady are conjunctivitis, photophobia and profuse secretion of tears with occasionally loss of vision. Such electrical flashes as cause eye troubles are usually due to short circuiting, loose electrical connections and strong arcs. Dr. Apfelbach cites 50 cases of electrica ophthalmia occurring in a large iron works. It is the actinic rays present in electrical flashes which do harm. Electricians exposed to accidental short circuiting often suffer from temporary blindness of 1 or 2 hours' duration. In some people the eyes become bloodshot and the eyelids swollen. Photophobia is a common symptom. Patients feel as if the eyes contained sand. Keratitis and iritis are not unknown. A similar remark applies to pigment changes in the retinæ. As a rule the prognosis is good. A more detailed statement of the harmful effects of exposure to electric light and to electric welding is given in my book "Dangerous Trades" by the late Mr. Simeon Snell of Sheffield. The application of a few drops of a 4 per cent. solution of cocaine to the eyes gives relief to pain. Irrigation of the eyes with weak boracic lotion may be necessary, followed later by the instillation into the eyes of a few drops of argyrol—25 per cent. solution. If photophobia continues, smoked glasses ought to be worn. Dr. Apfelbach states that in some of the large steel works electrica ophthalmia is prevented by the men wearing glasses made up of two blue glasses with a red one between. By this means the strong actinic or ultra-violet rays are prevented filtering through.

In all generating stations and in factories wherein electricity is used on a large scale, danger signals should be employed to avert the disasters alluded to in the text, while all workmen likely to be brought into contact either with live metal itself or with fellow-workmen injured by electrical contact should be instructed in rescue work and in the precautions necessary for self-protection.

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RESUSCITATION FROM ELECTRIC SHOCK

RULES RECOMMENDED BY

COMMISSION ON RESUSCITATION FROM ELECTRIC SHOCK

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NATIONAL ELECTRIC LIGHT ASSOCIATION

FOLLOW THESE INSTRUCTIONS EVEN IF VICTIM APPEARS DEAD

I. Immediately Break the Circuit

With a single quick motion free the victim from the current. Use any **dry** ***non-conductor*** (clothing, rope, board) to move either the victim or the wire. Beware of using metal or any moist material. While freeing the victim from the live conductor have every effort also made to shut off the current quickly.

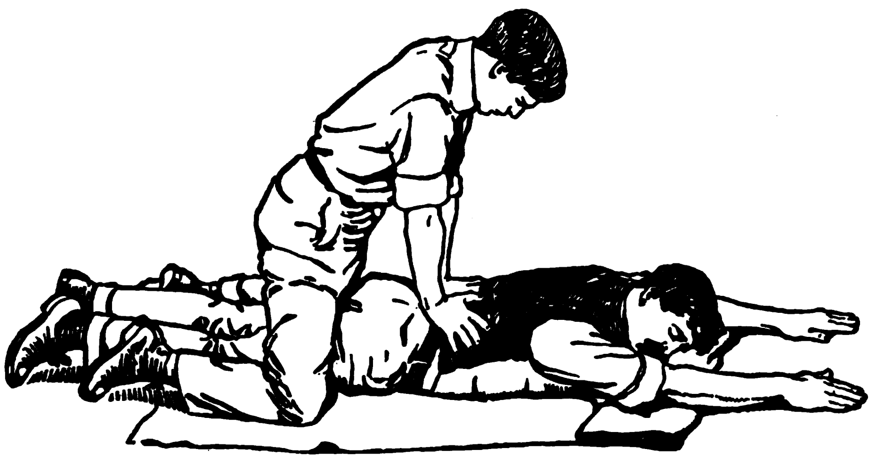

FIG. 27.—Expiration; pressure on.

II. Instantly Attend to the Victim's Breathing

1. As soon as the victim is clear of the conductor, rapidly feel with your finger in his mouth and throat and remove any foreign body (tobacco, false teeth, etc.). Then **begin artificial respiration at once.** Do not stop to loosen the victim's clothing now; **every moment of delay is serious.** Proceed as follows:

(*a*) Lay the subject on his belly, with arms extended as straight forward as possible, and with face to one side, so that nose and mouth are free for breathing (see Fig. 27). Let an assistant draw forward the subject's tongue.

(*b*) Kneel straddling the subject's thighs and facing his head; rest the palms of your hands on the loins (on the muscles of the small of the back), with fingers spread over the lowest ribs, as in Fig. 27.

(*c*) With arms held straight, swing forward slowly so that the weight of your body is gradually, but *not violently*, brought to bear upon the subject (see Fig. 28). This act should take from 2 to 3 seconds.

(*d*) Then immediately swing backward so as to remove the pressure, thus returning to the position shown in Fig. 27.

(*e*) Repeat deliberately 12 to 15 times a minute the swinging forward and back—a complete respiration in 4 or 5 seconds.

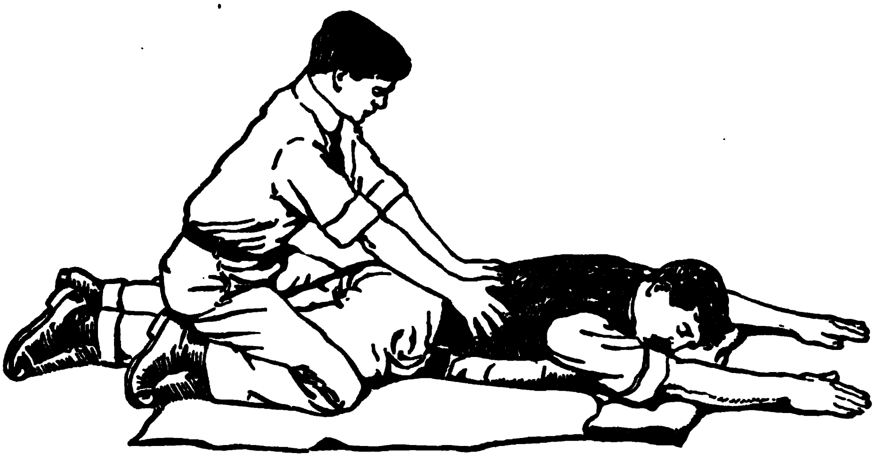

FIG. 28.—Inspiration; pressure off.

(*f*) As soon as this artificial respiration has been started, and while it is being continued, an assistant should loosen any tight clothing about the subject's neck, chest, or waist.

2. Continue the artificial respiration (if necessary, 2 hours or longer), **without interruption**, until natural breathing is restored, or until a physician arrives. If natural breathing stops after being restored, use artificial respiration again.

3. **Do not give any liquid by mouth until the subject is fully conscious.**

4. Give the subject fresh air, but keep him warm.

III. Send for Nearest Doctor as Soon as Accident is Discovered

The following Doctors are recommended:

Name	*Address*	*Telephone*	*Call*

The prone-pressure method of artificial respiration described in the rules (Section II) is equally applicable, after clearing the mouth and throat of froth, to the resuscitatoin of the apparently drowned, and also to cases of suspended respiration due to inhalation of gas or to other causes.

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PART II

ETIOLOGY AND PROPHYLAXIS OF OCCUPATIONAL DISEASES. VOCATIONAL HYGIENE

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Photograph supplied by Dr. Hanson through the courtesy of the American Telephone and Telegraph Company.

FIG. 29.—Anti-asphyxiator for supplying fresh air to cablemen while at work in the cable vaults, which are below the street surface. This is a mask which fits tightly over the nose and mouth, having two valves which work automatically as the man breathes; one valve supplies the fresh air as he inhales and the other carries out the vitiated air as he exhales. The intake is set up beside the manhole, pointed in the direction from which the wind is blowing, and connected to the mask by rubber pipe. Cablemen have been known to work every day for over a week in gaseous manholes with the aid of this device.

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DIVISION I

Etiology and Prophylaxis of Occupational Diseases

BY GEORGE M. KOBER, M. D., Washington, D. C.

Health is the chief asset of the workingman, and no greater calamity can befall him than when his earning capacity is impaired or arrested by reason of sickness or disability. It means in many instances the utter financial ruin of the family, and is doubtless one of the most potent causes of poverty and distress. Many diseases are incident to occupation and environment and industrial efficiency, and earning power can be promoted by appropriate safeguards and adequate protection of those engaged in gainful occupations.

Definitions.—Diseases of occupation may be defined as injuries and disturbances of health contracted in industrial pursuits, and other vocations in life, as a result of exposure to toxic agents, infectious organisms or other conditions inimical to health.

These diseases may be acute or chronic and vary in intensity and duration from the acute and fatal attacks of asphyxia, caused by suffocating gases, to the slow and insidious forms of industrial tuberculosis.

Classification.—An exact classification of occupational diseases is difficult, as we have as yet no standard nomenclature for this class of diseases, nor do they differ essentially in the majority of instances from the pathology and clinical picture of diseased conditions in general. For the present we may divide them into three general classes, viz:

(*a*) *Specific occupational diseases*, which include all diseases caused by industrial poisons or specific organisms which enter the system in the course of employment and are traceable to definite materials used, also diseases and sequelæ caused by exposure to increased atmospheric pressure.

(*b*) *Systemic occupational diseases* include all disorders caused by the movements, position of the body, overexertion, dust and fumes and abnormal working conditions, such as excessive heat, cold, moisture, or sudden changes.

(*c*) *Occupational diseases of special senses and organs* include all affections due to exposure to defective or intense light, excessive heat and noises, mental and physical strain, mechanical, toxic and caustic agents, etc.

But even a classification according to causes is not exact, as many of the diseases contracted in industrial pursuits are due to a combination of causes and the so-called predisposing factors play a very important rôle. We will now briefly consider the most important causes of occupational diseases:

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Industrial poisons, according to Dr. R. Fischer, are to be defined, in general, as those raw materials and products, by-products, and waste products which, in their extraction, manufacture and use in industrial processes may, notwithstanding the exercise of ordinary precaution, find entrance into the body in such quantities as to endanger, by their chemical action, the health of the workmen employed.

Eulenburg's Encyclopedia, published in 1898, under the heading "Arbeiterhygiene" gave a list of 31 industrial poisons. This list was amplified in 1908 by Prof. Th. Sommerfeld, Sir Thomas Oliver and Prof. Felix Putzey, and again amended by Industrial Inspector Dr. R. Fischer, in 1910. Sommerfeld's final draft was referred to the permanent advisory council of hygiene of the International Association for Labor Legislation, composed of Dr. R. Fischer of Berlin, L. Teleky of Vienna, J. P. Langlois of Paris, Sir Thomas Oliver of England, and Prof. Dr. L. Devoto of Milan, by whom it was edited, approved and finally published by the International Labor Office (Basle, December 24, 1911). A full translation of this list was published by the Bureau of Labor, in Bulletin 100, May, 1912. The list deals with 54 poisons. Because of its importance it is reproduced on page 720. The advisory council of hygiene of the International Association for Labor Protection also approved Fischer's definition of industrial poisons.

Acute and Chronic Industrial Poisoning.—The industrial poisons may enter the system through the respiratory organs, the digestive tract, through the skin or by a combination of these channels. The effects are either acute or chronic and vary in severity from the fatal attacks of acute chlorine or aniline poisoning to the chronic intoxications from arsenic, lead, mercury, phosphorus, etc. Acute forms of industrial poisoning are fortunately becoming less frequent, but many of the industrial poisons may produce both acute and chronic effects. In the acute forms we are dealing with profound functional disturbances caused by an overdose of the various blood and nerve poisons; in the chronic forms, because of diminished dosage, the poisons cause disease of a chronic character, by producing gradual histological changes of the blood and tissues of the body. There are doubtless numerous instances in which minute quantities of poison are not sufficient to produce specific effects, and yet bring about a general deterioration and diminished power of resistance, as instanced by the undue prevalence of tuberculosis among lead and mercury workers.

It is well to remember that, as in infectious diseases so in the occupational intoxications and systemic diseases, dosage, individual susceptibility, a low state of vitality, previous illness, bronchial and gastric catarrh, wounds and abrasions of the skin and the alcohol habit are important predisposing factors. Since most of the poisons are eliminated by the lungs, liver, kidneys and skin, functional derangement of these organs also plays an important rôle.

The selective affinity of industrial poisons for certain organs or tissues

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remains obscure. We can appreciate why lead should attack the nerves of the overworked muscles of a painter's right arm and cause paralysis, commonly called "wrist-drop," or why white phosphorus should show a special affinity for a carious tooth and aid the microörganisms in their destructive work in bringing about necrosis or the "phossy jaw" of match makers. So far, however, no reasonable explanation has been offered why "wood alcohol" should select the optic nerve, lead the nerve tissue, the tunica media of arteries, and arsenic chiefly the nerves of the lower extremities; why hysteria or neurasthenia should develop as a result of chronic poisoning from three such widely differing substances as carbon bisulphide, manganese and mercury. But after all many similar phenomena are found in diseases in general, and the predilection of disease germs for certain organs or tissues in particular awaits explanation.

The most important occupational intoxications are discussed in this work by recognized experts and this discussion is limited to a statement of general facts.

Prevalence of Industrial Poisoning.—The actual number of cases of industrial poisoning cannot be determined until there is a uniform law requiring physicians to report certain occupational diseases. In countries like England, where reports have been compulsory since 1897, it is quite possible to secure, for example, reliable data as to the number of cases of lead poisoning. The same may be said of the facilities offered by the statistics of the "German Industrial Insurance Institute," which furnish not only the number of deaths but also the number of cases treated, together with the age period and the duration of the disease. Similar data should be collected in all industrial countries. As a matter of fact, prior to 1910, in the absence of statistical information in this country there was a general impression that American workmen were largely immune from the dangers of industrial poisons. The publication of Dr. Andrews' report, based upon a study of 15 match factories, disclosed the fact that 65 per cent. of the employees were exposed to the dangers of phosphorus poisoning, and 82 cases of serious poisoning were found in three factories alone. This report and the investigations of Drs. Alice Hamilton and Andrews on the prevalence of lead poisoning in the United States, also conducted by the Bureau of Labor, together with the facts collected by the Illinois Commission on Occupational Diseases, led to a realization of the importance of full and accurate knowledge concerning the existence of dangers from industrial poisons and their prevention. Thanks largely to the efforts of the National Association for Labor Legislation, Congress, after two years' delay, by an Act approved April 9, 1912, practically abolished the manufacture of poisonous matches by imposing a tax at the rate of 2 cts. per 100 matches upon all white phosphorus matches manufactured in the United States after July 1, 1913.

In 1911 six states, California, Connecticut, Illinois, Michigan, New York, and Wisconsin, enacted laws requiring physicians to report certain occupa-

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tional diseases in their practice. In 1912 Maryland and New Jersey enacted similar laws, and during 1913 Maine, Massachusetts, Minnesota, Missouri, New Hampshire, Ohio, and Pennsylvania enacted similar legislation. The lack of uniformity in these laws is to be regretted. The most comprehensive law was enacted in Missouri. We have as yet no tabulated returns from these reports, and while it is doubtful whether even one-fourth of all the cases will be reported, the returns will at least furnish more accurate knowledge concerning the existence of industrial hazards from this source.

This knowledge will be of great practical value because, as a result of preventive measures, factory sanitation and education of the employees, a gratifying reduction in the morbidity and mortality of wage earners has everywhere taken place. For example, in Great Britain, where notification was made compulsory in 1897, the number of cases of lead poisoning has been reduced from 1278 in 1898 to 505 in 1910.

The economic importance of the subject must be apparent from the fact that according to the Leipsic Industrial Insurance statistics of 1910, 1270 cases of lead poisoning were cared for, with an average duration of 36.2 days, involving 45,794 days' loss of work. This is all the more lamentable since many of these patients are subject to relapses. The Vienna statistics cited by Koelsch[1] show that of 516 painters, during the course of 4 years, 263 suffered from one attack, 85 from two attacks, 25 from three, 1 from eight and 1 from nine attacks. According to Kaup's[2] statistics the morbidity rates among about 5000 Berlin painters between 1905 and 1908 were 20 per cent. above the average in other occupations, the duration of illness 80 per cent. higher, the mortality rates during the productive age 50 per cent. higher, and the tuberculosis death rate 4 per 1000 higher. The Leipsic statistics also reveal an undue prevalence of gout among the workers in lead trades.

The number of cases of mercurial poisoning has also been greatly diminished by the employment of less harmful substances and general precautionary measures. For example, the number of sick days from mercurialism, which in 1885 among 160 mirror platers at Fürth amounted to 5463 days, has been wholly eliminated by the substitution of nitrate of silver. The number of cases among the miners and smelters of Idria has been reduced from 122 in 1896 to 5 in 1908.

Similar gratifying reductions are reported in phosphorus poisoning, and with prohibitive legislation it is only a question of time when cases of necrosis of the jaw, which formerly afflicted between 11 and 12 per cent. of match makers, will entirely disappear.

According to Bauer,[3] 4320 cases of poisoning occurred in the German Chemical Industry between 1894 and 1904. From 1900 to 1905 the number of accidents was far above the average of 66 other occupations. In 1907 there were still 371 cases of poisoning in this industry, with 151 deaths. Of 170 cases from toxic gases 69 occurred in this industry, 32 were caused by

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carbon monoxide, 27 by furnace and illuminating gases, 16 by mine, powder and dynamite gases, and 2 by well gases. Fortunately the number of such accidents is constantly diminishing, especially in the aniline and coal-tar industry. In a roburite factory at Witten, with a morbidity rate of 96 per cent. in 1895, no cases of poisoning from dinitrobenzol have occurred since 1903.

Importance of the Industrial Poisons from the Medico-legal Standpoint.— The question of international protection of labor was agitated as early as 1870, but it was not until 1890 that the first international conference was held, in Berlin. This was followed by an International Congress at Paris in 1900 and the establishment of an International Association for Labor Protection, with a Bureau at Basle, in 1901. The object of this organization is to secure the enactment of uniform labor laws, to collect and publish literature on the cause and prevention of industrial diseases and accidents, and to promote the welfare of the laboring classes in general. It is largely due to the efforts of this organization that the use of white phosphorus in the manufacture of matches has to a great extent been prohibited and that so much has been accomplished in the prevention of lead poisoning, by the substitution of leadless processes in different industries. Indeed there are many leaders in the medical profession, men like Sir Thomas Oliver, Professor Sommerfeld and others, who believe that the interests of humanity demand the substitution of white zinc for the use of white lead in paint. It is also due to the efforts of this organization that men in the front ranks of social medicine prepared a list of industrial poisons. The care exercised, so that no injustice may be done to employees under the liability acts, is shown by Dr. Fischer's definition of industrial poison. The question whether the benefits of the workingmen's compensation acts, which formerly applied to accidents only, should be extended to cases of acute industrial poisoning came up, and a number of countries decided to include certain of the occupational diseases with industrial accidents. The question now uppermost in the minds of thoughtful men is whether it is a social justice to allow damages in the case of loss of a limb by machinery accident, and to ignore the claims of a workman whose arm is disabled by lead paralysis, and whether it is fair that the dependents of a wage earner who perishes from an attack of acute industrial poisoning should receive a pension, while those in the case of a worker who dies from the chronic effects of the same poison do not. Already France, Switzerland, and England have extended the original list of notifiable diseases. In England, for example, the Act of 1906 which originally covered only six industrial diseases was extended in 1907–1908 and again in 1913. It now includes the following:[4] anthrax, arsenic, ankylostomiasis, lead, mercury, phosphorus, nitro and amido derivatives of benzene, denitrobenzol and aniline, carbon bisulphide, nitrous fumes, nickel carbonyl, African boxwood, chrome, ulceration, ulcerations produced by dust or caustic or corrosive liquids, cancer or ulcerations of the skin or of corneal surface of eye due to pitch, tar or

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tarry compounds, chimney-sweeps' cancer, glanders, compressed-air illness or its sequelæ; and the following affections peculiar to miners: nystagmus, beat-hand (subcutaneous cellulitis) of coal miners, bent knee and bent elbow and inflammation of the synovial lining of the wrist-joint and tendon sheaths of miners; also cataract in glass workers, telegraphist's cramps, and writers' cramp. "In order to claim compensation a workman must produce: (1) a certificate from the local certifying factory surgeon; (2) or prove that he had been suspended from his usual employment on account of having contracted one of the diseases scheduled; and (3) in the event of death proof will have to be given, by friends or dependents, that death was due to one of the diseases mentioned above (Oliver)."[5]

It is not improbable that the extension of the liability act to dangerous occupations will involve more or less litigation and possibly also lead to the practice of malingering. There is already more or less literature on this subject, and also on the relation of a traumatic injury received to the development of diseases which, sooner or later, incapacitate the individual from work. Considerable evidence has been accumulated on chest injuries in relation to the development of pneumonia or to the lighting up of a latent tuberculous lesion of the lung, on injuries of the knee as an exciting cause of tuberculous inflammation of the knee-joint, and on blows upon the abdomen as a cause of Addison's disease. Traumatic injuries in general are regarded as an occasional cause of malignant growth, and the same is true of habitual exposure to aniline, coal tar, paraffine, etc. Diabetes insipidus may develop as a result of injury to the back of the head, and diabetes mellitus is liable to be aggravated, if not induced, by severe physical and mental strain.

The German Industrial Insurance system appears to reduce the chances for litigation to a minimum by securing perfect coöperation between the employer, the employees, technical experts, factory inspectors, officials and physicians.

Measures for the Protection of Industrial Workers against the Dangers of Poison; Compiled by Industrial Councillor Dr. Fischer of Berlin.

"1. Properly adapted buildings, thick walls of separation for dangerous rooms, good lighting, facilities for keeping the workshops clean and for effective ventilation.

2. Apparatus adapted to its special purpose, whenever possible, closing tight in every part.

3. Appliances for accomplishing the arrest of gases and dust at their place of origin, their removal (by exhaust fans), and in a suitable manner rendering them innocuous or collecting them, thus preventing their entrance in the nose and mouth.

4. So far as possible, avoidance of direct contact with poisonous materials or substances injurious to health in working with them, transporting or packing them.

5. The displacement of particularly dangerous labor methods and mate-

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rials by the introduction of less dangerous labor processes and material, as well as by the employment of materials satisfactorily pure chemically.

6. Instruction of workmen, just entering upon an occupation, concerning the properties of the poisonous substances extracted, manufactured, used or otherwise evolved, and, whenever possible, cautionary leaflets should be placed in the hands of the workers.

7. The repetition of these instructions at frequent intervals.

8. Posting of precautionary regulations and warning placards, containing admonitions for the exercise of special caution, and enjoining the observance of measures for insuring safety. Constant supervision of all dangerous employments by expert and responsible persons.

9. Employment of appropriate means for personal protection in the way of suitable work clothes, caps, gloves, goggles, and, as necessary adjuncts, mouth and nose shields, respiratory masks and the like, in case the appliances named in rule 3 are inapplicable.

10. Practice of bodily cleanliness by the use of wash, bath and dressing rooms, the use of special rooms for eating, separate lockers for street and work clothes, and frequent non-hazardous cleaning of the clothing.

11. Immediate report of symptoms of indisposition, attention to wounds of the skin caused by the handling of corrosive materials, prompt employment of a reliable antidote, and summoning at the same time of a physician.

12. The employment of a healthy working force. Periodical medical examination of the workers in dangerous employments. Temporary or permanent exclusion of unfit workmen from the dangerous departments of the industry. Under certain circumstances there should be a change of work in occupations giving rise to chronic poisoning.

13. The utmost possible reduction of the hours of labor in dangerous employments."

Many of these excellent rules will be extended under specially hazardous employments. It is needless to point out that every dangerous industry should be provided with a "first-aid outfit." In employments involving exposure to toxic gases or electricity, oxygen apparatus, pulmotors and Meltzer's resuscitation apparatus are indispensable.

Rescuing parties must likewise seek protection in the way of forced ventilation, breathing helmets, etc. It is cheaper, and certainly more humane, for large concerns to provide not only these safeguards, but also competent medical service, than to pay compensation under the liability act. But after all the chief aim and object must be prevention.

Industrial Infectious Diseases.—In this class of diseases the germs enter the system in the course of employment and are traceable to the materials used or to the soil, premises or environment. The most important infections, such as anthrax, uncinariasis, tuberculosis, etc., are dealt with in special chapters and also under different occupations, hence only a few salient facts bearing on general etiology and prevention will be presented here.

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(*a*) *Uncinariasis*.—This infection is liable to occur among all workers in fecally polluted soils, especially in miners, tunnel workers, quarrymen, brick and pottery workers, farmers and planters and persons employed in lumber and construction camps. The economic importance of hook-worm infection must be apparent when, according to Ashford,[6] about 800,000 persons, or 90 per cent. of the laborers in the coffee and sugar plantations of Porto Rico, were infected. Stiles[6] estimates the number of victims in the United States at 2,000,000, and the economic loss from inability to work at $100,000,000 per annum. That this disease is both preventable and curable is shown by the results achieved in Porto Rico and in certain mining districts of Europe (see pages 181, 624).

(*b*) *Anthrax*.—This occupational infection, from the standpoint of frequency, is insignificant when compared with the hook-worm disease. The prevalence of anthrax in different occupations is well illustrated by the German statistics of 1910, covering 287 cases with 39 deaths and affecting 257 males and 30 females. According to Koelsch,[7] in 135 cases the infection was traced to live or dead animals, in 142 instances to the offal of infected animals, one case was contracted by a laboratory worker, and in nine instances the source of infection could not be determined. The 257 male cases were distributed as follows: Agriculture, 121; flaying establishments, 9; hide trades, 8; tanneries, 92; leather, 3; transport laborers, 7; horsehair factories, 11; brush and hair factories, 4; wool carding, 1; and glue factories, 1.

(*c*) *Actinomycosis*.—This disease, known as "lumpy jaw" in cattle, is caused by the ray fungus, and is generally believed to be transmitted from animal to man. Since the natural habitat of the fungus is on grass and grain straw, it may possibly be conveyed by placing grass or grain straw in the mouth. The majority of cases occur among farmers and stockmen. At all events, of the 27 cases studied by Rigler at the clinic of Jena, 18 affected farmers. The disease is characterized by chronic, generally localized, suppurative conditions, accompanied by destructive processes of the tissues, and if the seat of the lesion affects some internal organ the termination is usually fatal.

(*d*) *Foot and Mouth Disease*.—This affection primarily affects cattle sheep, goats, and pigs and is communicable to man. The disease, also known as "aphthous fever," is occasionally observed in persons connected with the animal industries. After an incubation of from 4 to 5 days the disease is ushered in by a chill, followed by fever and the formation of vesicles upon the tongue, lips and mucous membrane of the buccal cavity and pharynx. The vesicles soon break and leave painful ulcers. Occasionally also miliary and pustular eruptions are observed on the hands.

(*e*) *Glanders and Farcy*.—This disease affects primarily the horse, ass, or mule and may be transmitted to man through wounds and abrasions, by direct contact with the mucopurulent secretions, by droplet infection, by the inhalation of dried and pulverized discharges, or through the medium of

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horse blankets, harness, stable utensils, etc. The disease is also communicable from man to man. It is known as glanders when it attacks the mucous membranes and as farcy when it affects the skin. In either case the course may be acute or chronic and may vary in severity from a slight local infection to a profound general pyæmic condition. The prognosis is extremely unfavorable, as from 50–80 per cent. of the cases terminate fatally. Bollinger, cited by Koelsch, has collected 106 cases; of these, 66 occurred in hostlers and coachmen; 10 in veterinarians; 6 in horse butchers and 6 in persons employed in flaying dead animals; 5 in soldiers; 4 in physicians; 2 in horse dealers; 1 in a horseshoer; 1 in an anatomical laboratory worker; and 5 in other occupations.

(*f*) *Septic infections* are not infrequently observed, especially among persons engaged in the flaying of dead animals, the manufacture of artificial manure, glue and tallow factories, the hide, tanning and rag industry, in renovating hair mattresses and feathers, in silk workers, cannery employees, butchers, game dealers, cooks, physicians, veterinarians, laboratory men, workers in aniline, grease, paraffine, etc. The streptococci and staphylococci may enter the system through wounds and abrasions, through the medium of biting flies or insects, or through the sebaceous follicles, or they may be inhaled. The infection may manifest itself by localized affections, such as furuncles, or by the graver forms of cellulitis, lymphangitis, erysipelas, septic sore throat and joint infections, and even by profound general septicæmia.

(*g*) *Smallpox* and *cowpox* are occasionally contracted by persons engaged in handling infected clothing, laundry, bedding, rags, feathers, etc. The danger of handling fresh feathers and raw cotton imported from countries where smallpox is endemic has recently been emphasized by Shablowsky[8] and by Corbin.[9] The disease may also be contracted by nurses and attendants, and by employees of hotels, inns, etc., especially by chambermaids. I recall two small epidemics in which the infection was evidently carried all the way from Chicago by a traveller who, although apparently well himself, left foci of infection in two hotels.

Cowpox is occasionally contracted by farmers and dairymen, especially during the act of milking.

(*h*) *Syphilis.*—The virus of the disease caused by the "spirocheta pallida" evidently clings to the secretions of syphilitic ulcers and may be conveyed in an extragenital way by kisses, wet nursing, use of infected pipes, cigars and cigarettes, by the promiscuous use of glass-blower's pipes and the mouthpieces of wind instruments, by dental and surgical instruments, and during digital examination by physicians and midwives, in case of abraded surfaces, hangnails, etc. Scheuer,[10] who collected 297 cases of occupational syphilis, mostly among physicians, midwives and nurses, also reports a series of cases in paper-hangers, cabinetmakers, shoemakers, saddlers, painters, dressmakers, who were infected by putting in the mouth or between the lips such

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things as pencils, nails, awls, needles, wire, thread and brushes, previously infected by syphilitic fellow-workers. According to the same author, clerks, accountants, and waiters have contracted the disease by holding infected lead pencils, pen holders, and even coins between their lips; he also reports instances of transmission in persons employed in the laundry and rag industries.

(*i*) *Tetanus* is a typical soil disease and infections have been reported as incident to occupation, not only in workers of the soil but also in the jute industry.

(*k*) *Tuberculosis.*—This disease, on account of its importance, is discussed in a separate paper. It is not a specific occupational disease, except perhaps in some instances where the infection is conveyed in autopsy work, or in the transmission of bovine tuberculosis, as in the case of butchers, cooks and dairy employees, who have contracted tuberculosis of the skin by handling infected meat, milk and cream.

The writer,[11] in 1903, collected 12 cases of tuberculous wound infection in veterinarians and butchers, and three cases of accidental inoculation in man by the topical application of cream and milk. Lassar[12] reports 11 cases of verrucose tuberculosis in butchers and abattoir employees from wound inoculations. It is also conceivable that the disease is occasionally conveyed by infected clothing and foot wear; at all events Perlen[13] informs us that of 4177 tuberculous subjects treated in the Münich clinic 709 were engaged in repair and cleaning shops of foot wear and clothing.

There is abundant statistical evidence, both here and abroad, that persons engaged in the mechanical and manufacturing industries pay a terrific toll to the so-called "white plague." Our latest census statistics, from the registration area in the United States, show that the industrial classes, which constitute about one-third of the population, contribute almost one-half of all the deaths from tuberculosis. It is now generally held that in nearly 90 per cent. of the poorer classes, tuberculosis is contracted in childhood and the germs remain dormant until adverse conditions create a favorable soil for their growth and development. Such a soil is usually found in persons whose body has been weakened from any of the numerous causes which exist, whether it be a previous attack of sickness, malnutrition, loss of sleep, vice and dissipation or other debilitating factors.

In view of the fact that, as a general rule, only able-bodied persons enter the ranks of industrial workers, the question naturally arises, why should such a large percentage of strong men and women fall victims to the disease? This is not at all strange when we consider the many unfavorable factors to which they are subjected, such as crowded and insanitary workshops, long hours in a bad air, overwork and fatigue, deficient light, dampness, exposure to extremes of heat and cold, sudden changes in temperature, and last but not least the inhalation of dust, toxic fumes, etc. All of these factors are calculated to lower the power of resistance and favor not only the

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development but also the spread of the disease, especially when some of the workmen are already afflicted and are careless in the disposition of their expectoration.

The influence of occupation is strikingly shown by the fact that the tuberculosis rate among 472,000 males in the United States, exposed in 15 occupations to the inhalation of organic dust, was 2.29 per 1000, against a rate of 1.55 for all occupied males; and also by the fact that 42.05 per cent. of the deaths of printers, lithographers and pressmen, who died at the ages between 25 and 44 years, were from consumption, as compared with 21.85 per cent. for farmers, planters and overseers. Similar data collected by Koelsch in Bavaria, Elben in Würtenberg and the Registrar General of Great Britain could be adduced to emphasize this point. Suffice it to state that the German Industrial Insurance statistics show that while only 77 agriculturists per 1000 are invalided by consumption, the rate among industrial workers is 245.

Hirt was one of the first to show that persons employed in dust-producing occupations suffer much more frequently from pneumonia and consumption than those not exposed to dust and that there is practically no difference in frequency of disease of the digestive organs.

	Consumption	Pneumonia	Digestive disorders
Workers in metallic dust	28.0	17.4	17.8
Workers in mineral dust	25.2	5.9	16.6
Workers in mixed dust	22.6	6.0	15.2
Workers in animal dust	20.8	7.7	20.2
Workers in vegetable dust	13.3	9.4	15.7
Workers in non-dusty trades	11.1	4.6	16.0

Perlen[13] analyzed the histories of 1426 tuberculous patients with reference to dust exposure and found that 30 per cent. had been exposed to metallic dust, 26 per cent. to vegetable dust, 18 per cent. to mineral dust, 17 per cent. to mixed dust, and 8 per cent. to animal dust.

Sommerfeld's statistics show that, with an average tuberculosis death rate of 4.93 per 1000 of the population in Berlin, the rate in non-dusty trades was 2.39 and in dusty trades 5.42. In trades giving rise to *metallic dust*, 5.84; in *copper*, 5.31; *iron*, 5.55; *lead*, 7.79. In trades giving rise to *mineral* dusts, *pottery* workers, 14; *masons*, 4.26; *stone cutters*, 34.9. *Organic dusts*, *leather*, *furs* and *feathers*, 4.45; *wool* and *cotton*, 5.35; *wood* and *paper*, 5.96; tobacco dust, 8.47.[1]

The amount of dust is perhaps less important than the character and chemical composition of the particles composing it. For this reason, no doubt, the hard, sharp, and angular particles of iron, steel, and mineral

[1]See pages 484, 504, 520, 579, 585, 608, 617, 642, 656, 659, 665, 669, 678, 683, 701, 777.

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dust are more liable to produce injuries of the respiratory passages, thus favoring the invasion of bacilli or lighting up latent lesions. On the other hand, it will be shown in subsequent pages that exposure to certain kinds of dust, such as coal, lime, gypsum, sulphur and tan bark, may have an inhibitory effect on the disease.

It would be unfair not to consider the influence of home environments such as unclean, crowded or otherwise insanitary dwellings, insufficient or improper food, ignorance or disregard of correct living and working conditions, and, last but not least, the bad effects of the abuse of alcohol. It has been shown that the abuse of alcohol not only lowers the general resistance of the body, but the habit of visiting and remaining in saloons for hours, sometimes till midnight, deprives the individual of proper rest and exposes him to devitalized air and the toxic fumes of tobacco, carbon monoxide and dioxide gases, and other injurious agents. The influence of alcohol is well demonstrated by Guttstadt's statistics. He found that the average death rate from tuberculosis in Prussia was 16.1 per cent. of all the deaths, the rate in the liquor trades was 22.3 and among bartenders and waiters as high as 52.6 per cent. But as pointed out by Koelsch the question of physique plays an important rôle, since the rates among the robust brewers and malt workers of Bavaria, in spite of their intemperate habits, is only 3.25 and 3.56 respectively. Vice and dissipation is not confined to wage earners and the startling facts remain that, even with due consideration of all the contributory causes referred to, the rates from industrial tuberculosis are excessive. Indeed it has been estimated that over 70,000 wage earners perish every year from this disease in this country.

Preventive Measures.—It is perfectly safe to state that, by effective methods for the prevention and removal of dust, fully one-half of these lives could be saved.

This is not at all speculative, since Prof. Röpke informs us that, as a result of general education and factory sanitation, including of course cuspidors and the prevention of promiscuous expectoration, the mortality at Solingen, in Germany, the population of which is largely made up of employees in the cutlery industry, has been reduced from 20.63 per 1000 in 1885 to 9.3 per 1000 in 1910, and the consumption death rate from 540 in 1885 to 180 per 100,000 of population in 1910. Similar data are available to show that diseases of the respiratory organs in one of the German cement works have been reduced from 9.3 per cent. to 3.3 per cent., after the installation of a suitable apparatus for the removal of dust.

What can be done in some of the most dangerous industries in Germany can be done in this country, and will be done as soon as the importance of the subject is fully appreciated.

Röpke's table, comparing Solingen with Sheffield, which is the seat of the cutlery industry in England, is reproduced for the lesson it conveys.

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MORTALITY STATISTICS

	Solingen Industrial Insurance Statistics 1909–1910*			Sheffield Med. Officer of Health, 1901–1909†		
	Total mortality per 1000	Mortality from tuberculosis	Other diseases of the respiratory organs	Total mortality per 1000	Mortality from tuberculosis	Other diseases of the respiratory organs
Grinders, polishers, buffers, etc.	9.30	4.37	2.35	30.40	15.10	5.40
Other employees in the steel and cutlery industry	9.00	2.66	1.79	29.30	5.80	6.90

The marked contrast between these two cutlery centers was commented upon by Mr. C. Johnston, in the Annual Report of the Chief Inspector of Factories, as early as 1906. We quote from this report as cited by Oliver as follows: "The atmosphere of Solingen is bright and clear. It is seldom that black smoke is seen escaping from the factory chimney. This is largely the result of careful firing and the use of coal briquettes, instead of ordinary coal. The factories are said to be marvels of order and cleanliness; the floors are of concrete, and the air space for each worker is 565 cu. ft. All the grinding stones are protected by guards. The walls of the work rooms are lime washed every year; the floors are swept clean every evening and damp wiped once a week. The 'racing' of grindstones is never undertaken during the working hours, except under a stream of water, or unless the stone is entirely inclosed in casing, except at the working place of the raising tool. One of the features of Solingen is said to be the large number of 'home workers.' These workmen own their own houses, behind which is a garden and at the rear of this is their small factory. There are 1475 small factories of this type in the district. In these the greater part of the grinding and glazing of scissors and razors is done. The floors are kept clean and provisions are made for the removal of dust during the grinding. Cutlery manufacture is recognized as a dangerous trade in Solingen, and in recent years considerable improvement has taken place in the means to prevent dust. With the clean and tidy appearance of these tenement factories those of Sheffield compare most unfavorably. In Solingen the grindstones and polishing wheels are run toward the worker; in Sheffield they are run away from the worker so that the dust has an upward tendency and flies into the room."

(*l*) *Parasites.*—Among the parasites transmissible from animals to man should be mentioned cysticerci or the larval form of the tapeworm,

* Based upon the statistics of 7908 grinders and 10,872 other employees in the metal industry.

† Based upon 3941 grinders and 3889 other metal workers.

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and infection with trichina spiralis, which is occasionally acquired by butchers, sausage makers, fishermen and cooks, mostly as a result of the habit of eating raw beef, pork or fish.

Scabies and pediculosis are not infrequently contracted in lumber camps. Bedbugs and fleas are also great pests in unclean camps. The body louse may transmit the virus of typhus fever. The *pediculoides ventricosis*, described by Schamberg and Goldberger, occurs primarily in wheat; it persists in the straw for months and may possibly survive in flour. The eruption caused by this parasite resembles either a severe form of urticaria or erythema multiforme. It is liable to affect farm hands, harvesters, packers of the straw and all who may later come into contact with the straw, and possibly those who handle the flour (Hazen). Among other mites capable of setting up troublesome forms of dermatitis are the mower mite, harvest bug, and chicken lice, which chiefly affect farmers, gardeners, poultry raisers and feather workers.

The barbed hairs of the *brown-tailed moth*, and of its cocoon and caterpillar contain an irritant poison, which not infrequently causes a dermatitis, resembling urticaria, in persons engaged in the eradication of the pest.

Lumbermen, rivermen, guides and other field workers frequently suffer, not only from the local effects of mosquitoe bites, but also from systemic infections such as malaria, yellow fever, etc. They are not infrequently exposed to the bites of moose and deer flies, sand flies or chiggers, etc.

Injurious Environments.—There is abundant evidence to show that the baneful effects attributed to occupations are in large part caused by faulty environments and working conditions and hence to a great extent avoidable.

Indoor Occupations and Bad Air.—It is generally held that indoor employment is inimical to health, while out-door work in a pure air favors health and longevity. This statement appears justified in the light of statistics furnished by Körösi, who found that out of 1000 deaths 436 occurred in persons engaged in indoor occupations against 322 employed in out-door work. The rate in England (with a standard of 100) was 55 for fishermen, 61 for gardners, 62 for agriculturists, against 84 for grocers, 152 for cloth dealers, 144 for tailors and 233 for printers. The latest U. S. statistics show that the mortality from tuberculosis was 16.1 per cent. in agricultural pursuits and 23.1 per cent. among bookkeepers and accountants, 23.7 per cent. in tailors and 28.2 in printers, lithographers and pressmen. If we stop right here in the comparison the evidence would be overwhelming in favor of out-door employment. But when we find that the mortality from consumption among merchants and dealers (except wholesale) is only 16.1 per cent., exactly the same as in agriculturists, and for draymen, hackmen, and teamsters it is 23.3 per cent., almost the same as for bookkeepers, it becomes apparent that in estimating the hazards of indoor occupations other factors, such as physique, habits, exposure to dust, social conditions, standard of

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living, etc., must be considered. One of the chief dangers of indoor life is exposure to vitiated air. The air of dwellings and workshops is never as pure as the outer air, because it is polluted by the products of respiration combustion and decomposition. The presence of individuals also tends to vitiate the air with dust, germs and organic matter, from the skin, mouth, lungs and soiled clothing. Unless provision is made for the dispersion of foul air, and the introduction of pure air there is much reason for assuming that these impurities play a more or less important rôle in what has been designated as "crowd poisoning," characterized in the acute form by symptoms of oppression, headache, dizziness, and faintness, while the chronic effects of deficient oxygenation and purification of the blood are plainly evinced by pallor, anæmia impaired appetite, and gradual loss of physical and mental vigor. All of these effects are intensified by exposure to excessive temperature and moisture, especially when human beings are obliged to occupy a space with an air supply insufficient for the proper oxygenation of the blood. As a result of habitual exposure to vitiated air we note an undue prevalence of consumption and pneumonia in crowded workshops, dwellings, prisons, public institutions, and formerly also in military barracks and battleships. Overcrowding naturally favors contact and droplet infections from tuberculosis, pneumonia, influenza, septic sore-throat, etc. The influence of overcrowding on diseases of the air passages, amounting at times to epidemics, was well illustrated on the Isthmus of Panama and, as suggested by General Gorgas,[14] accounts probably for the undue prevalence of these diseases among the gold miners of the Transvaal. By scattering the laborers on the Isthmus from large and crowded barracks into single huts and small rooms, with not less than 50 ft. of floor space, the pneumonia rate was reduced in a single year from 18.4 per 1000 to 2 per 1000, and in urging a similar procedure for the Rand he predicts a like reduction. Another bad effect of indoor occupations is that the work is usually performed in a sedentary and stooped position, which, apart from interfering in youthful workers with the full development of the chest, limits expansion of the lungs and also causes constipation, congestion of the portal circulation and hemorrhoids.

The baneful effects of vitiated air are of course intensified when the occupation is attended with the production of dust and fumes, the foes of industrial life.

Cubic Air-space and Amount of Fresh Air per Hour.—It is evident that the question of cubic air-space for each worker plays an important rôle and for the following reason: It is known that carbon dioxide is not itself a toxic agent, but an excess of this gas in the air of rooms leads to a deficiency of oxygen, and also to defective elimination of CO_2 from the system, which cannot be eliminated, whenever the tension of CO_2 in the air exceeds that of the carbon dioxide in the blood. In order that the respiratory impurities may not exceed certain limits (6 volumes of carbon dioxide per 10,000) it has been found that an average adult requires 3000 cu. ft. of fresh air per hour, and

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this amount should be supplied without discomfort to the occupant. Experience has shown that the air of a room cannot be changed oftener than three times in 1 hour in winter without causing a disagreeable draft, hence every occupant should have cubic air-space of 1000 ft. This is the ideal standard, and one of the factory laws of New York (1906), relating to certain manufactures in tenements, provides that the whole number of persons therein shall not exceed one to each 1000 cu. ft. of air-space. Such an ideal standard is not always obtainable in workshops and it is believed that for practical purposes, an air-space of 500–600 cu. ft. per capita will suffice, provided the air is renewed sufficiently often.

A number of states require an air-space of 250 cu. ft. for each employee, between the hours of 6 A.M. and 6 P.M., and, unless by written consent of the factory inspector, not less than 400 cu. ft. between the hours of 6 P.M. and 6 A.M., provided each room is lighted by electricity. This is a step in the right direction, but it would be extremely desirable to place the minimum amount of cubic air-space at 500 ft. for day work and 600 ft. for night work, unless electric lights are used, in which case a uniform standard of 500 ft. might be prescribed. At all events the question of sufficiency ought not to be left to factory inspectors. Either the cubic air-space should be specified, or what is equally effective the carbon dioxide contents of the air should be limited to 12 volumes per 10,000. The authorities of Great Britain have made a beginning in the latter direction by placing the limit of CO_2 at 20 volumes when gas or oil is used for lighting (or within 1 hour thereafter) and 12 volumes when electric light is used (or within 1 hour thereafter) and 9 volumes per 10,000 at any other time.

Ventilation, which means the removal and dispersion of bad air and the introduction of fresh air, is accomplished either by natural or artificial means. Natural ventilation is usually sufficient when each occupant has 1000 ft. of cubic air space; the walls of the building are porous or contain numerous crevices near the doors and windows, the difference between the indoor and outdoor temperature is considerable, and the winds strike the walls directly or pass with great velocity over chimney flues and other openings. But as the direction and force of the winds, and the other factors referred to, cannot be controlled, other means should be provided for ventilation. For this purpose open windows, doors, and revolving fans answer very well in summer. The objection to this method is the cold drafts in winter. In rooms heated with direct radiation the fresh air should therefore be admitted above the heads of the occupants, either by fresh air register inlets in the walls or by the insertion of louvered or swinging windows. Thus an upward direction is given to the air, so that it may impinge, on the ceiling, mix with and be warmed by the heated air in this situation, falling gently into all parts of the room, being gradually removed by means of foul air outlets, aided by exhaust fans. Another simple plan is to bore slanting holes in the bottom rail of the window sash, or to employ a Pullman or Bury ventilator, or to insert a

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piece of board 4 in. wide across the window sill. The separation of the sashes thus caused will provide for indirect fresh-air inlets.

Artificial ventilation, may be secured by providing: (1) suitable inlets and outlets; (2) by extraction by heat, or the creation of a decided difference between the inner and outer temperature; and (3) by propulsion and aspiration. Space will not permit to enter into details except to say that, besides the contrivances already mentioned, any of the ordinary registers in which the air passes through the walls by means of a perforated iron plate, and is then directed upward by a valved plate with side checks, will prove of service. McKinnell's ventilator consists of two cylinders, one inside the other and of different lengths; the longer tube, projecting above and below, serves to conduct the impure air, while the outer cylinder, having a larger sectional area, serves as an inlet. The outlet is protected on the top by a cowl, and both tubes can be regulated by valves. They are especially useful in the ventilation of one story buildings, or the upper story of any building. If gas is used as an illuminant, the burners may be placed immediately under the extracting tube. As the warm air escapes through the inner tube a corresponding volume is admitted through the interspace between the two cylinders.

Ridge ventilators consist of openings through the ceilings and roof, with louvered sides and ends, protected by a small roof. The opening of the air shaft in the ceiling is usually provided with suitable registers. The fresh air is admitted by the means already referred to, or by registers placed behind radiators. If the building is heated by stoves, the fresh air may be admitted by inlets running underneath the floor, between the joists, and discharging through a register near the stove.

Extraction of foul air by heat is usually accomplished by placing a separate flue next to the chimney flue. The latter, if in use for firing purposes, creates an upward current. If this is not sufficient it may be promoted by gas jets or a steam coil placed in a separate flue.

The propulsion and aspiration system is especially adapted for all large buildings and factories, and consists of mechanical devices by which the fresh air is forced into and distributed throughout the building by the use of fans or air propellers. The foul or objectionable air is removed by so-called exhaust fans, preferably placed near the floor.

Temperature and Humidity.—It is a well-known fact that the welfare and capacity for work of individuals are to a great extent influenced by the surrounding temperature.

There are numerous occupations involving exposure to extremes of heat and cold, dampness and sudden changes, the effect of which will be briefly stated. The human organism possesses the faculty of maintaining a uniform temperature, *i.e.*, it so regulates and harmonizes the production and the loss of animal heat that the normal temperature of the blood, 98.2°F. is not materially affected. In this the skin doubtless plays the most important rôle. Whenever cold acts upon the skin the irritation is primarily exerted upon the

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nerves which transmit it to the central organs of the nervous system (the heat regulating center) and from there it is reflected to the nerves of the cutaneous vessels and muscular fibers, which promptly contract, and in consequence of a diminished blood supply there is less loss of heat. If, on the other hand, heat instead of cold acts upon the skin, we have dilatation instead of contraction of the vessels, with an increased surface blood supply, and corresponding loss of heat by radiation and conduction. At the same time the perspiratory glands are stimulated to greater activity; more sweat is excreted and evaporated, and still more heat is dissipated. One of the bad effects of profuse perspiration is that the blood is deprived of some of its constituents. The blood is taken away too long from the internal organs, the proper distribution of the blood supply is interferred with, and in consequence the tone and nutrition of the stomach, lungs, heart, and other internal organs is lowered. We lose our appetite and suffer from indigestion, the red corpuscles are decreased, we experience languor and general enervation, and the system in consequence is rendered more susceptible to disease.

While the human organism endeavors to adapt itself to extremes of heat and cold, the facility of the body to maintain the equilibrium is by no means unlimited, and the heat-regulating center is liable to fail, or become paralyzed if imposed upon too long or too frequently. This is especially the case during sudden changes of temperature. It is the abruptness which offends the peripheral nerves, and the greater the abruptness the more intensive will be the irritation which is transmitted by reflex action to other parts of the body, usually the weakest parts. This may result in driving the blood to internal organs, causing congestions and other mischief. Then again a cold draft, playing on the skin, may cause neuralgia, paralysis, sore-throat, bronchitis, or pneumonia, showing that cold applied locally may excite diseases in the neighborhood of its application or in distant organs, and finally it may produce disease by checking the secretions of the skin.

Humidity.—The atmosphere always contains a certain amount of water in the state of vapor, which varies from 30 per cent. to complete saturation, or, according to temperature, from 1 to 12 grains in a cubic foot of air. The degree of atmospheric humidity is of special hygienic importance, as it influences to a great extent the cutaneous and pulmonary exhalation of vapor and, in consequence, also affects the animal temperature. The average daily amount of water eliminated by the skin is 2½ lb. and about 10 oz. by the lungs. It is evident that when the air is damp it lessens evaporation, as it possesses little drying power, and the water from the skin and lungs is with difficulty evaporated. The evaporation of perspiration, by which heat is rendered latent, is one of the chief means of cooling the body. Consequently when the air is hot and moist the humidity tends to increase the effects of the heat, the blood is with difficulty kept at its proper temperature, and all the disagreeable effects of a high temperature are intensified. This condition may be so aggravated that the temperature of the body exceeds the

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normal degree and causes our cases of heat stroke or heat exhaustion, which occur especially on hot, sultry days.

A damp, cold, or chilly air also produces mischief, as it abstracts an an undue amount of animal heat, lowers the general vitality of the system, and favors the development of diseases of the respiratory passages, neuralgic and rheumatic affections, and aggravates the severity of such attacks. We may conclude, therefore, that excessive humidity tends to intensify the effects of both heat and cold. On the other hand, excessive dryness of the air is also harmful. It increases evaporation, the skin becomes dry and chapped, and the mucous membranes of the mouth, eyes and respiratory passages are irritated, causing catarrhal conditions.

Exposure to excessive heat is especially common in stokers and firemen, smelters, puddlers, blast and electric furnace men, steel mill and foundry men, blacksmiths, glass-blowers, kiln and pottery men, bakers, cooks, miners, tunnel workers, malsters, piano polishers, soldiers, sailors, roofers and all out-door workers during hot weather. Exposure to dry heat for a short period is sometimes borne without serious effects in temperature as high as 140° or 150°F., provided the air is kept in motion, and a temperature of 212°F. may be borne by puddlers and electric furnace men, for a very brief period, without serious injury. Prolonged exposure, however, is usually followed by grave constitutional disturbances already referred to. Apart from heat exhaustion, which is not especially common, we see more frequently as acute manifestation, cases of colic, concentrated urine and muscular cramps, which symptoms are more or less influenced by toxins generated within the body; cases of "colds," anæmia and general debility are also quite common in this class of workers. The "colds" are due to the pernicious habit of the employees in passing suddenly from an overheated atmosphere to the outer air during the cold months. As already stated, any abrupt change in temperature is liable to cause congestion of internal organs; hence the undue frequency of catarrhal, neuralgic and rheumatic affections among imprudent workers. These congestions not infrequently also result in gastro-intestinal and vesical catarrh, and pave the way for pneumonia, pleurisy and Bright's disease. The men engaged in handling molten metal, and all others exposed to *radiant* heat, not infrequently suffer from dermatitis and pigmentation of the skin, inflammatory conditions of the eyes, and even cataract, probably induced by a partial dehydration of the tissues. Nervous affections, such as headache, dizziness and general irritability, are also observed. Exposure to intense *solar heat* is not uncommon among out-door workers, and cases of sunburns and sunstroke occasionally occur. Sunstroke is evidently caused by a congestion and inflammation of the cerebral membranes, in which not only heat but the ultra-violet rays may play a rôle. *Heat combined with humidity* is especially trying to workers in breweries, laundries, kitchens, hot houses, tanneries, canneries, and sugar refineries, but cases of heat exhaustion may occur in all occupations involving

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hard work in a hot and sultry atmosphere. Affections of the skin, such as cystic degeneration of the sweat ducts, are not uncommon in laundry workers and others exposed to a steaming atmosphere.

The effects of exposure to *excessive cold* are usually not so serious, as they are generally guarded against by suitable clothing and food rich in fats and carbohydrates. The persons chiefly affected are ice men, cold storage workers, butchers, brewery men, in the cooling department, and out-door workers such as lumbermen, aviators, farmers, teamsters, coachmen, etc. In this instance, also, any sudden change of temperature is liable to engender internal congestions. Among the local effects may be mentioned frost bites and eczema.

Occupations involving exposure to a *cold and humid atmosphere* are injurious, because a cold damp air abstracts an undue amount of animal heat from the body, lowers the power of resistance, and predisposes to catarrhal, rheumatic and pulmonary diseases.

Preventive Measures.—The most agreeable temperature for average healthy adults performing ordinary light work is between 65 and 70°F., but a lower temperature is desirable for persons engaged in hard work. Every effort should be made to avoid extremes of heat and cold. Much may be done to reduce the temperature of workshops by forced ventilation and a supply of cool fresh air. The windows should be kept open and the air kept in motion. Special cooling devices and screens have been designed for blast furnaces, etc.

Since an average relative humidity between 55 and 65 per cent. has been found most healthful, efforts should be made to maintain such a standard whenever practicable. Apart from methods calculated to accomplish this result, reliable thermometers and hygrometers are required for efficient control. Legislators would do well, instead of making a general provision for "sufficient heat, moisture, etc., to prescribe a standard, at least in industries where such standards are practicable and can be reasonably enforced.

The English regulations for the textile industries prescribe that the temperature shall not fall below 50°F. and "the humidity of the air shall not at any time be such that the difference between the readings of the wet- and dry-bulb thermometers is less than 2°."

Many of the injurious effects of exposure to extremes of heat and cold can be guarded against by suitable clothing, avoidance of abrupt changes, frequent bathing and systematic hardening of the skin.

Abnormal Atmospheric Conditions.—The effect of compressed air upon the workmen in caissons and diving apparatus have been presented in the chapter on caisson disease, and the effects of rarified air upon mountain climbers, baloonists, aviators and workers in high altitudes have been described on page 211. The effects of air concussion and excessive noises have been considered in the chapter on diseases and injuries of the ear.

Abnormal Light Conditions.—The effects of excessive or defective light

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are set forth in the chapter on diseases and injuries of the eye, and it remains for us to emphasize here the principles which should govern the subject of lighting.

Natural Light.—The natural light in workshops should be sufficient, so that the eyes need not be strained even on cloudy days. When the light is defective the objects have to be brought too near. The eyes in consequence converge. The muscular strain thus produced causes a gradual elongation of the anterior-posterior axis of the eyeball, and nearsightedness results. It is believed by specialists that 80 to 90 per cent. of the headaches are due to eye strain caused by defective light, also numerous cases of backaches, anæmias, and general impairment of health. In addition there is good reason to believe that defective lighting influences the number of industrial accidents, the efficiency of production, the quality of the product and the cleanliness, cheerfulness and healthfulness of the shop.

It has been found by Putzeys[15] that the natural lighting in temperate climates will usually come up to hygienic requirements when the area of windows, exclusive of sash frames, equals one-sixth of the floor space. In order that the light may penetrate the deeper portions of the room, the windows should reach almost to the ceiling and the glass should be either pure white or prismatic, and kept clean.

The difficulty of securing a sufficient amount of daylight in buildings located in narrow streets, surrounded by tall buildings, has been partly overcome by glass building blocks 8 by 6 by 2½ in., with an air chamber in the center, used instead of bricks or stone, in connection with steel-frame construction, but more particularly by the introduction of prismatic glass, which refracts and diffuses the light.

Artificial light, no matter how obtained, differs from daylight in this, that it does not furnish a pure white light, the prevailing rays being red, yellow or violet. Whatever difference of opinion there may be as to the color best suited to the eye, we know that vision is most perfect under the influence of white light, and this ought to be a good criterion. One of the disadvantages of all low-power illuminants is that the light is never as bright as daylight, involving, therefore, closer application of the eyes and consequent strain of the muscles of the eyeball. These remarks are hardly applicable to the electric arc light and the Welsbach gas-burner, the rays of which, like the direct solar rays, may indeed be so glaring as to cause undue irritation of the retina.

Another harmful effect of artificial illumination is the unsteady or flickering character, especially seen in the electric arc light, which on account of the abrupt changes is likely to irritate the retina. Another disadvantage is that the ordinary illuminants, except the electric light, tend to vitiate the air by the products of combustion, and also affect the temperature and humidity of the air by the heat evolved.

The requirements of a hygienic light are that it should be as near as

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possible the color of the sunlight, sufficiently ample but not too glaring. It should be steady, and instead of deteriorating the air it should as far as practicable be utilized to promote ventilation; nor should the heat evolved be sufficiently intense to be a source of discomfort to the inmates in warm weather. The most common methods of lighting now employed are the electric incandescent lamps, arc lights, mercury-vapor lights, electric bulbs, gas light and kerosene lamps. Of these, electric light, especially the indirect system of lighting, is superior to gas or other illuminants, because there is little or no danger from fire, and there are no products of combustion, hence no pollution of the air. Nor are the temperature and humidity of the room affected to any perceptible extent. These advantages over gas or kerosene are of special importance to the inmates of buildings where the question of fresh air and temperature plays an important rôle. Hence many industrial plants find it profitable to install the very best type of electric lighting, and thereby save time and money by the prevention of sickness and accidents among their employees. Next to the electric light, gas, especially in connection with a Welsbach or Siemen's burner, or acetylene gas, offers the best choice. In the absence of either electric or gas light, kerosene, with a high flashing point, should be preferred over other illuminants. Suitable outlets for the products of combustion should always be provided.

White, clean ceilings and walls will be of great service, not only in solving the question of light but also in general sanitation. A number of States require the walls to be limewashed or painted.

The sufficiency of artificial lighting may be approximately determined by observation, and quite accurately by the employment of Bunsen's method and his photometer. In this country and in England, according to Munson, "the unit adopted for the measurement and comparison of lights is a No. 6 sperm candle burning 8 grams per hour and giving out a light known as 1 candle-power." Such a candle contains on analysis carbon 80 per cent., hydrogen 13 per cent., oxygen 6 per cent., and in combustion yields to the air equal volumes of carbonic acid and watery vapor, namely, 0.41 cu. ft.

Dust and Fumes.—The relation of dust to occupational diseases is extremely important and its effects upon the respiratory organs have been considered by Sir Thomas Oliver in the Chapter on Pneumokoniosis. The desirability of differentiating fibroid phthisis, according to the nature of the foreign particles or exciting cause, has given rise to special designations. So, for example, fibroid phthisis caused by coal dust has been called *anthracosis*, by flinty or silicous particles, *chalicosis* or *silicosis*, from clay dust *aluminosis*, from steel, iron or iron oxide, *siderosis*, from tobacco, *tabacosis*, from cotton or linen dust, *bysinosis*, and from feather and down dust (Watkins-Pitchford[16] has recently coined the term) "*ptilosis*" with special reference to the ostrich feather industry in South Africa.

Ahrens[17] found the amount of dust per cubic meter of air in different establishments as follows:

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	Mg.		Mg.
Horse-hair works	10.0	Flour mill	28.0
Sawmill	17.0	Foundry	28.0
Woolen factory	20.0	Foundry polishing room	71.7
Woolen factory with exhaust ventilation	7.0	Felt-shoe factory	175.0
Paper factory	24.0	Cement works	224.0
Laboratory	1.4		

Dr. Graham Rogers[18] found 70 grains of dust per million liters of air in a skirt factory, about the same amount in a pearl button factory, and 75 grains in the air of a brass foundry.

According to Schuler and Burkhardt, cited by Roth,[19] the morbidity per 1000 workers in dusty trades is as follows:

Bookbinders	98	Paper-factory employees	343
Silk weavers	205	Mechanical industrial shops	419
Cotton spinners	250	Wood turners	427
Cotton weavers	285	Rag sorters in paper mill	429
Typefounders and typesetters	304		

Dust, apart from its relation to respiratory diseases, also plays an important rôle in diseases of the eye, ear, nose and throat, as instanced by an undue prevalence of chronic inflammatory conditions of these organs in lime, cement and hair workers, and by the frequent occurrence of ulcerations of the nasal septum in chrome, chlorine and cement workers. It has also been shown that even flour and sugar dust, usually considered quite free from danger, may be converted into lactic acid in the mouth and possibly increase the virulence of disease germs, as evinced by an undue prevalence of caries and pneumonia in flour and sugar workers. The dust generated in the manufacture of pearl buttons, from the shells of certain mussels, is liable to produce a peculiar form of osteomyelitis, involving especially the long bones of youthful workers, and other affections noted under mother of pearl workers. A combination of dust, sweat and heat, also favors the development of skin diseases, as seen by the undue prevalence of furuncles and eczema in persons exposed to mineral, metallic, sugar, flour, aniline and other dusts. Since dust and germs often go hand in hand, there is little doubt that in many instances occupational infections are conveyed by means of infected dust.

The effects of smoke and soot upon the health of workers and the community has been studied by Ascher of Germany, and by numerous antismoke commissions both here and abroad. The results everywhere show a distinct relationship between the smoke nuisance and diseases of the respiratory organs, especially pneumonia.

The injurious effect of dust in all its forms cannot be questioned and is made sufficiently apparent by a study of tuberculosis in relation to dusty trades. Fortunately only about 25 per cent. of the dust inhaled actually

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reaches the lungs, the bulk, according to Lehmann and his pupils, is swallowed. But this does not imply that the dust which has found its way through the stomach remains harmless, for it may reach the lungs by way of the lymph channels, and certainly all poisonous dusts can be absorbed in the gastrointestinal canal.

In addition to the mechanical, irritant and toxic character of dust, Haldane very properly points out that "it inevitably tends to lower the social status and self-respect of the work people."

Preventive Measures.—The subject of dust prevention, and its removal when its formation is unavoidable, is one of the most important problems. Thanks to the efforts of technical experts, most commendable progress has been made in the following directions:

(*a*) *Prevention of Dust Formation.*—There are a number of occupations in which dust production can be reduced to a minimum by the application of oil, sprays of water, or jets of steam. The wet processes are especially applicable to the metal, lead and pottery industries, to rock drilling, mining, blasting, stone crushing, and cutting, etc. Dr. Watkins-Pitchford,[20] in discussing the etiology and prevention of phthisis among the Rand miners, with a notoriously high morbidity rate, viz., 32 per cent. and a death rate of 13.8 per 1000, tells us that in December, 1912, there were 5600 rock drills in operation. He describes in detail the present methods of drilling and how the dangerous quartzite dust, as soon as it is formed, is converted into mud by keeping the drill hole and surroundings wet with water. This may be done by means of a syringe or spraying apparatus, while some types of rock drills provide for the automatic flushing of the hole. In blasting operations similar good results, in laying dust, have been attained by the application of water spray, or steam, just before the charge is fired. This is not only important in the prevention of dust inhalation, but also to prevent disastrous explosions of coal dust in coal mines.

Dr. Watkins-Pitchford[20] declares, "It is no mere euphemism to say that a scrupulous obedience to the spirit of the present Mining Regulations will result in the total abolition of pulmonary silicosis from the gold fields of the Rand." General Gorgas in a more conservative way says, "I advise that such measures be generally and carefully enforced and extended. I believe they will soon reduce miners' phthisis to a minimum." See also miners, page 622.

(*b*) *Prevention of Dust and Fume Diffusion.*—Technical experts have done much to prevent the escape of dust and fumes at the point of origin, by enclosing the machinery or apparatus in air-tight cases. This, however, prevents the diffusion of dust and fumes only when no opening or hand feeding is required, and hence the attempt to substitute as far as practicable automatic feeding machinery for hand labor. There are, however, many processes in which wholly enclosed machinery is impracticable and ineffective, and hence additional methods must be invoked.

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(*c*) *Removal of Dust and Fumes.*—This is accomplished by mechanical devices connected with a system of exhaust ventilation. Fortunately quite a number of states have made statutory provisions, for the removal of dust and fumes, which lay down specific rules concerning the construction of work benches and hoods or hoppers. The hoods are usually made of metal and so arranged and applied, that the dust will be thrown by centrifugal force into the hood and carried off by the current of air into a suction pipe attached to the hood or hopper. The suction pipes attached to different hoods enter a main suction pipe at an angle of 30–45°. The main or trunk line is usually below the floor and empties into a discharge pipe, connected with an exhaust fan, run at such a rate of speed as will produce a velocity of air sufficient to carry off the dust and fumes. It is extremely important that the mechanism is so arranged, that the dust and fumes are drawn away from the face of the worker, *i.e.*, downward and backward. The details of an efficient exhaust system must be worked out and adapted to the needs of different industrial plants, by a competent ventilation engineer.

(*d*) *Collection of Dust and Fumes.*—In the interest of public health and for economic reasons, in certain industries, the dust and fumes extracted by the preceding methods should be collected, treated and disposed of in a suitable manner. In the case of toxic fumes and gases this is usually accomplished by (1) condensation, (2) absorption by water or chemicals, (3) destructive distillation by heat in a closed vessel, (4) combustion of gases that can be burned, and (5) discharge of gases into the air at great height.

The material recovered by condensation and absorption constitutes often a valuable by-product of the industry. The value of blast-furnace and coke-oven gases is evinced by their utilization as fuel for heat and power, and the sulphurous fumes evolved in spelter works are converted into sulphuric acid.

In the case of dust which has no special value, it is usually made to pass through a tower and precipitated by means of a fine spray of water. For the recovery of valuable constituents, as in the jewelry industry, the dust is collected in suitable tanks under water for the ultimate recovery of gold or silver. The collection of large particles of dust is usually accomplished by means of so-called cyclone separators in which, by means of centrifugal force and a cone-shaped metallic drum, the dust is deposited.

A very efficient method is filtration of the air, through jute or cheese cloth, woolen sheets or fine wire mesh. The filter devised by Beth is supplied with a mechanical knocking device "which shakes the dust from the screening material to the bottom of the casing, where a worm automatically carries it to the collecting receptacle."

In addition to these methods successful attempts have been made to precipitate dust and smoke by strong electric currents. A method devised by Cottrell, in connection with some cement works in Southern California, has demonstrated that, by means of a large electrified chamber, from 95 to

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98 per cent. of the cement dust, from escaping gases during the roasting process, can be precipitated and collected.

(*e*) *Respirators.*—There are times when, in the absence of proper safeguards, or because of unavoidable working conditions, the employment of respirators becomes necessary. There is an endless variety of these devices in the market, all intended to filter out dust and fumes, by means of sponge, cotton wool or gauze. It should be remembered that even the best respirators, so far designed, are far from satisfactory, and none fulfil the indications fully. Indeed some of the respirators improvised by the workers, consisting of a piece of sponge, cheese-cloth, jute, etc., fastened by means of elastics over nose and mouth are quite as effective as most of the manufactured appliances.

The working conditions should be so perfect as to obviate the use of respirators. Until this is accomplished their employment should be encouraged.

(*f*) *Cleaning of Workshops.*—A number of states provide by law that "all factories shall be kept clean." It is difficult to set up exact standards, for what is clean in a blacksmith shop would not be clean in a shirt-waist factory. But it is well to emphasize the fact, that no amount of ventilation can do away with the necessity for frequent and systematic shop cleaning. Hygiene demands that there should be no accumulation of dust in any part of the premises, and therefore condemns all interior finishes, such as exposed girders, cornices, mouldings, cubby holes, unnecessary shelves and inaccessible spaces, which will serve as dust and germ traps.

Hygiene, on the other hand, approves of hard wood or impermeable floors, curves instead of corners and angles, smooth and non-absorbent walls—in brief of everything which will prevent the collection of dust and germs and facilitate their removal.

Workshops should be swept daily after cessation of work, the sweeping should be done when practicable with damp saw dust, with the upper windows opened. In certain industries, cleaning and dusting by the vacuum system has been practised and should be encouraged, especially where there is exposure to poisonous dust. All work benches and tables should be carefully cleansed, and wiped with a damp cloth for the removal of fine dust. Feather dusters do not remove but simply displace dust. In a number of occupations, cement floors, with a suitable incline for drainage, so as to facilitate washing with hose, have been found very useful. The application to floors of so-called "dust oil" cannot be approved since it simply allays dust, but does not remove it, and is therefore especially objectionable in industries where poisonous dust is given off.

Miscellaneous Sanitary Provisions.—In this connection it is desirable to point out certain sanitary requisites, which are important in all dusty occupations, especially in those involving exposure to toxic dust and fumes.

1. *Suitable Work Clothes and Caps.*—There is a great variety of suitable patterns in the market, of which the snug-fitting duck union suit, without many folds, properly buttoned and adjusted is the best. Such suits and

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caps should be furnished at the expense of the employer and washed once a week. In occupations involving contact with poisonous liquids, rubber gloves and impermeable overalls or aprons offer the best protection. For all wet processes impermeable clothing or aprons should be worn. Asbestos clothing has been recommended for firemen, etc., but its weight is objectionable, and light leather suits or aprons are preferable. Ordinary work suits may be rendered practically non-inflammable by chemical treatment.

2. *Dressing-rooms, Lockers, Bath and Wash Rooms.*—It is desirable, in all dusty occupations, that the workmen should take off all their street clothing before beginning work, and this is absolutely essential when the occupation involves exposure to poisonous dust. For this purpose suitable dressing-rooms, provided with lockers for street suits and separate compartments for overalls, are necessary. Facilities for washing and bathing, brushes, soap and individual towels should be furnished. In most of the civilized countries statutory provisions have been made for these sanitary requisites, in all establishments in which poisonous substances are manufactured or used, and the result has been most beneficial. It is important that workers should be instructed to wash their mouths and teeth before eating and upon cessation of work. Indeed it is a good habit to rinse the mouth before swallowing even water, as much of the injurious dust lodged in the mouth will find its way into the stomach.

3. *Pure Drinking Water and Lunch Rooms.*—Hard work and dusty occupations produce thirst, and hence the necessity of an ample, easily accessible pure and cool supply of drinking water. Sanitary fountains are coming more and more in evidence in factories and workshops. The health and safety of employees exposed to industrial poisons demand that no food shall be taken, or tobacco in any form used, in the workrooms. A commendable number of establishments have provided special lunch rooms in connection with their dressing-rooms where either their own food can be warmed up, or rolls, sandwitches, coffee, tea, milk, soft drinks, fruit juice and hot soups may be obtained at a nominal cost. Coffee and tea allay thirst and are stimulants, without the depressing effect of alcohol and their use has materially lessened the evils of intemperance. Milk is especially recommended for workers in the lead and aniline industries and is often supplied free of charge.

Abnormal Positions of the Body.—The effects of a constrained working position, combined with a sedentary life, have been briefly mentioned in connection with indoor occupations. The effects are especially harmful in youthful workers whose osseous system is not fully developed and there is little doubt that most of the bone and joint deformities are developed in the earlier years of their work, and aggravated by habit. Among the more important should be mentioned the hollow chest and round stooped shoulders, caused by a stooped and cramped position, as seen especially in tailors, engravers, lithographers, watchmakers, metal grinders, shoemakers, and all others obliged to assume a more or less bent-over posture. In shoemakers (cobblers) the

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pressure of the last against the breast bone, aggravates the anterior compression and often causes a typical depression of the sternum. All thoracic postural deformities naturally interfere with free expansion of the lungs, and hence with the respiratory functions. A stooped or bending posture also interferes with the proper distribution of the blood supply, and invites congestions of the abdominal and pelvic organs.

As a matter of fact, a large number of this class of artisans show a peculiar predisposition to consumption, many suffer from anæmia, constipation, dyspepsia, and hemorrhoids, and the majority have a low average duration of life.

Round shoulders and lateral curvature of the spine are quite common in bearers of burdens, and in all occupations involving the elevation of one shoulder above the other, so that even a clerk by a faulty position, or in the absence of an adjustable chair, may acquire lateral curvature of the spine. Such deformities are also quite common in blacksmiths, locksmiths, cabinetmakers and others, largely because of faulty posture and workbenches, and also on account of the unequal development of certain muscular groups.

Among youthful workers, especially apprentices of bakers barbers, waiters, nurses, etc., "flat-foot" "knock-knee," and "in-knee" and varicose veins of the lower extremity are frequently observed, as a result of being on their feet too long. Varicose veins, eczema, and ulcers are also quite common in motormen, conductors, machine tenders, and others who are obliged to be standing the greater part of their working hours. Shultes[21] found varicose veins in 12.2 per cent. of German recruits, who had pursued an occupation involving a standing position, 4 per cent. in those who stood and walked, and 1 per cent. in those engaged in a more sedentary position, and none in those whose occupation was wholly sedentary.

Abnormal position combined with pressure is responsible for muscular cramps, sciatica, and neuralgic affections. The so-called "housemaid's knee," which is an enlargement of the patellar bursæ filled with fluid is notoriously common, not only in scrubber women, but in floor finishers, carpet layers, and others who are obliged to be upon their knees for a considerable time. This same factor also plays an important rôle in the causation of the miners "beat knee," which is a subcutaneous cellulitis over the knee-cap.

Preventive Measures.—Experience has shown that many of these deformities can be prevented by teaching apprentices proper posture. Indeed in view of the fact that so many of these physical defects disqualify men for military service, it seems desirable that this training should be begun in the school. This can be done by the universal use of adjustable seats and desks, correction of faulty positions, and exercise of opposing groups of muscles. In the factory or workshop adjustable seats and proper workbenches are of equal importance, and all persons engaged in occupations involving constrained attitudes should be encouraged to engage in well

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regulated gymnastic exercises. A number of states make seats for females obligatory; there is no good reason why this provision should not be extended to include men, whenever it is practicable. For the prevention of "housemaid's knee" suitable knee pads should be used.

For the relief of varicose veins and prevention of leg ulcers, massage, rubber bandages, or elastic stockings are indicated.

Flat-feet.—Captain J. R. Harris of the Medical Corps U. S. A. has written an excellent article[22] on this subject and concludes that the prime cause of flat or weak feet is improper posture and gait. For the correction and prevention of flat-feet he recommends the following:

"The habitual position in standing should be the position of attention, except that the heels should be 1 or 2 in. further apart than the toes. This is a position of muscular tonicity and readiness in which the position of the astragalus and the integrity of the arch are maintained.

The stride is changed from the usual waddle gait to the proper gait by teaching the Indian stride until the reflex centers in the lumbar cord shall have learned the new routine, usually a matter of a few lessons only. Then reduction of the exaggeration is permitted, and a set of corrective and strengthening exercises prescribed such as the following:

1. Rise upon the tip toes as high as possible and lower the heels again slowly.

2. Raise the toes as high as possible and relax.

3. Raise the body by pinching or pressing downward with all the toes, an action similar to "grasping" with the hands, endeavoring not to use the leg muscles.

4. Cross the right foot in front of the left and sway, carrying the weight alternately from the left to the right foot, with active pushing of the toes. Reverse the feet and repeat the exercise.

5. Stand with the left foot in the hollow of the right knee and swing about balanced on the right foot. Change and balance on left foot. Continue each exercise from 5 to 10 minutes according to the effect upon the muscles. Repeat all the above several times each day.

These exercises are best taken in barefeet. It will be sufficient in many cases to stand with the toes turned well in, draw up the arches and then carefully turn the toes out to the parallel position, holding the arches and ankles in position, and frequently repeating the maneuver. In all cases it is necessary to remember that the ankles must be held up over the foot, and the "slump" of ankle and out consciously combated.

The prejudice against intoeing of children should be strongly combated, since slight intoeing is the natural and strong position; and the exaggerated position or pigeon toe is simply a natural effort to conserve arches and muscles not normal in strength."

Dr. Harris supports Munson's objections to patent devices intended to support the foot arch, upon the ground that they relieve the symptoms of

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discomfort at the expense of making the underlying cause of the latter much worse, for they splint and restrict the use of the very muscles upon the development and strengthening of which the regaining and preservation of the foot arch depends.

Dr. Jay W. Seaver an eminent orthopedist and for many years Director of Physical Culture at Yale considers Dr. Harris's training suggestions for the prevention and correction of this deformity, eminently practical and in accord with the mechanics of the anatomy involved.

Overexercise of Certain Parts of the Body.—The causes and diseases due to the excessive use of certain muscles, to eye strain, and excessive noises are fully discussed in the chapters on Occupational Neuroses, and Diseases of the Eye and Ear. These affections are of the utmost economic importance, since they impair not only the efficiency, but also the earning power of the worker, and not infrequently compel a change of work.

Among the affections due to the overuse of the voice should be mentioned chronic inflammatory conditions of the pharynx and larynx, in public speakers (clergymen's sore-throat), singers, train announcers, auctioneers, street criers, etc. In some instances, laryngeal spasm and paralysis of the vocal cords, with complete loss of voice, have been recorded.

Constant local pressure, friction, and overuse of the arms and hands, is productive of much harm. This is seen in the formation of painful callosities in the palms and fingers, inflammation of the synovial lining of the wrist-joint and tendon sheaths, and enlarged bursæ. These conditions not infrequently are followed by septic infections, such as subcutaneous cellulitis, as seen in the "beat hand" and "beat elbow" of miners. Cysts of the wrist tendons have also been observed, in the Government Printing Office, in persons engaged in making firm pressure with the hand in folding heavy paper. Contracture of fingers and atrophy of the palmar aponeurosis are also frequently observed, as a result of hard manual labor.

Occupations involving constant jarring of the entire body are conducive to the development of neurasthenia, insomnia, and gastro-intestinal neurosis.

As an example of overstrain of certain groups of muscles should be mentioned the distended, yet flabby condition of the cheek, as observed in glass-blowers and performers on wind instruments. In these cases there is at first a hypertrophy of the muscular fibers, followed later by a partial atrophy which not infrequently results in actual rupture. The air forced into the salivary ducts causes dilatation and emphysema of Steno's duct, which is occasionally followed by rupture and painful affections of the parotid gland.

Emphysema of the lungs is in some instances the result of overstrain of the pulmonary tissue, as witnessed by the occurrence of such cases among glass-blowers, users of blowpipes, and performers on wind instruments.

Illustrations of the acute and chronic effects of excessive muscular strain are seen in the undue prevalence of cases of hernia, among persons engaged in hard work, especially in those who lift or carry heavy weights on ladders,

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as in the building trades. Fortunately derricks and lifts are diminishing these hazards. The effects of habitual hard work in the causation of diseases of the heart and circulatory system, which in some instances may result in sudden death from cardiac exhaustion or rupture of the blood-vessels have been pointed out on page 239. Indeed there is ample statistical evidence to justify the conclusion that persons habitually engaged in hard work are more frequently subject to disease and accident and present a higher mortality than persons more favorably situated.

Overwork or Fatigue.—One of the most important predisposing causes to disease is overwork or fatigue, because the accumulation of waste products in the blood, from muscular wear and tear, together with the expended nervous energy, combine to render the system more susceptible to disease. The subject of fatigue in its physiological and pathological relations is discussed by Professor Lee in a special chapter.

From the view point of prophylaxis it may be well to bear in mind the following maxims:

1. Life without daily purposeful effort is wrong; there are those who do too little work, and those who do too much. Some groups of muscles are overworked and others are not sufficiently used. The brain worker lacks physical exercise, and the physical worker mental exercise and training (Fisher).

2. Habitual hard work and long hours are calculated to diminish the general power of resistance and to bring about premature physical deterioration.

3. The more intensive the work ("speeding up") and the shorter the interval of rest for the elimination of waste products, the earlier we may expect manifestations of fatigue.

4. Excessive heat and humidity, exposure to vitiated air and toxic dust and fumes, violent concussions of the air, and general jarring of the body, constrained posture, and overexertion of certain muscles, favor premature fatigue.

5. Insufficient or improper food, long walks to work, vice and dissipation and the abuse of alcohol produce premature fatigue.

6. Monotonous work, employments involving mental strain and responsibility, watchful care in machine work, fear about satisfying the "boss," anxiety about money and domestic affairs are all conducive to premature fatigue, while work done with zest often acts as a tonic.

7. Night work constitutes a fatigue factor only when it involves long hours and loss of sleep.

"*Speeding Up.*"—Among all the fatigue factors, none is more potent than the pernicious practice of "speeding up." In order to meet the demands of competition, lower prices, high profits, and trade supremacy, inventive genius is ever at work to increase the speed and output of machinery, and employees have to keep pace with the machine. This speeding up is manifest

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in all of the mechanical industries, especially in the textile and clothing industry. Some of the sewing machines now carry ten needles instead of one, involving correspondingly increased strain of the eyes to watch for broken threads, and also increased nervous tension and physical fatigue. This high-pressure system had its origin in this country and is by no means confined to individual industries. The very excellencies of our progress have stimulated the nerves and intellect and fired the ambition of men until they overleap the limits of their natural powers. Rest and recreation seems impossible to many, and the temporary stimulant derived from the tempting cup offers, for the time being, relief to our physical and mental exhaustion. It is, however, like all so-called "nerve tonics," a dangerous remedy. It is like applying the whip to a tired horse and the result is a constant increase in the number of prematurely worn-out workers, neurasthenics, alcohol and drug habitues.

Neurasthenia is by no means confined to mental workers. Of 285 cases treated by Petren, 189 belonged to the laboring and agricultural classes. It is true that certain occupations involving exposure to industrial poisons, such as coal gas, carbon disulphide, lead and manganese, predispose to this disease, as do also occupations involving exposure to excessive heat, light and noise. But when we see an increase all along the line of industrial workers, affecting, according to Schwab, about one-fourth of the garment workers in St. Louis, it is evident that the foundation is chiefly laid in the "speeding up system," which has also invaded Europe.

Preventive Measures.—The general effects of overwork and chronic fatigue are characterized by loss of appetite, anæmia, digestive derangements, respiratory and cardiac affections, fatigue neuroses, neurasthenia, and general deterioration of health.

In the prevention of the far-reaching consequences it is evident that work must be regulated by the capacity of the individual, and hence it is desirable at the outset for persons to select only employments for which they are physically fitted. After entering upon employment it is important that all the fatigue factors should be carefully considered and controlled by wise and humane measures. So, for example, a change of work to less arduous tasks may often prevent a complete breakdown. It is obvious that there must be proper intervals for rest and recuperation, hence the necessity of regulating the hours of labor and the enforcement of a day of rest. We have referred on page viii to the high mortality of the industrial workers of Great Britain at the beginning of the nineteenth century. This was at a time when employees in the cotton mills were obliged to work from 14–18 hours a day. Koelsch informs us that after the reduction of the hours of labor, from 12–8 hours a day, in the alkali works of Norwich, the morbidity rate sank from 10.12 per cent. to 6.1 per cent. After the introduction of the 9-hour shift in the English machine shops, in 1872, the average span of life of these workers was lengthened in the course of 17 years from 38¼ to 48¼ years. In esti-

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mating the general efficiency of such commendable laws it is but fair to concede that the general health movement in the last four decades, especially the improvement of the air we breathe and the water we drink, has played an important part in the prolongation of human life.

The physiological remedy for fatigue is rest and sleep, which offers an opportuity for recuperation (see sleep, page 472).

Hours of Labor.—The enactment of laws limiting actual labor to 8 hours a day finds ample support in a study of the relation of fatigue to accident liability (see page 452) and can also be defended upon general health principles. This is especially true of females, who, on account of their imperfectly developed muscular system and more delicate physique, require special protection.

No child should be permitted to work in factories and wage-earning occupations under the age of 14, and then only upon presentation of a medical certificate that it is free from physical defects. Such children should not be obliged to work longer than 6 hours, with 2 hours of interval of rest after the first 3 hours. Under no circumstances should they be permitted to perform night work. The same may be said of individuals between the ages of 16 and 18 years, who, however, may be permitted to work 8 hours a day, with proper intervals for meals and rest.

Employment of Women and Children.—In the face of many adverse circumstances under which labor is often performed, it is but natural that the immature male employees and females should suffer most. The former not infrequently inherit a weak constitution, or acquire it by insanitary home surroundings and deficient food. Quite a number are obliged to enter upon active work long before their bodies are sufficiently developed. Apart from the fact that child labor is a menace to education, morals and good citizenship, the effects of premature and involuntary labor upon the health and physical welfare of the child are extremely detrimental. It has been shown that up to the age of 13 to 14, the muscular fibers contain a large percentage of water, and in consequence are very tender and weak. Demetjeff, cited by Rubner,[23] determined the lifting powers of the arm and trunk of the working class, and found that the average boy of 14 possesses about one-half the muscular strength of an average adult between 35 and 40 years.

LIFTING POWER OF THE ARM AND TRUNK OF THE WORKING CLASSES AT DIFFERENT AGES

Age	Kilograms	Age	Kilograms
14 years	82	30 to 35 years	150
16 years	101	35 to 40 years	160
18 years	128	40 to 50 years	148
20 to 29 years	140	50 to 60 years	134

As a result of imperfect development of muscles and bones it is not surprising that a large percentage of young persons engaged in workshops, factories, or even at the writing desk, develop lateral curvature of the spine and other

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deformities, such as flat-foot, knock-knee, in-knee, etc., not to mention general weakness, and a predisposition to other diseases. All of the bad effects are naturally intensified by insanitary environments, especially when the occupation involves exposure to dust and fumes. In quite a number of European countries minors are excluded from trades involving exposure to industrial poisons. During the last Census there were 1,752,187 children under 16 years of age engaged in gainful occupations; of these over 80,000 were employed in the textile industry; about 25,000 in mines and quarries; 12,000 in the manufacture of tobacco and cigars; over 10,000 in wood industries; 7116 in the glass industry; over 7000, mostly girls, in laundries; 2000 in bakeries; 138,000 as waiters and servants; 42,000 boys as messengers; and 20,000 boys and girls in stores.

According to Koelsch[24] women possess only from six-tenths to seven-tenths the strength of men of like weight, and yet, in the struggle for existence, quite a number enter the ranks of industrial workers. For physiological reasons they are quite sensitive to the effects of physical and mental fatigue, and it is perhaps fortunate that, because of marriage or inability to withstand the strain of modern industries, the majority abandon their strenuous work before permanent injury is done. But even as it is, Roth tells us that 75 per cent. of 145 female inmates in a German sanitorium suffered from anæmia, chlorosis and neurasthenia, as a result of overwork. It is a matter of common observation that women who have had to deny themselves proper rest and care during the menstrual period, for several weeks before and after confinement, are very liable to suffer from chronic uterine diseases and hemorrhages, while miscarriages and premature births are not infrequent results of overwork. Statistics collected by Neisser indicate that such accidents are quite common among farmers' wives, laundresses, seamstresses and women employed in the jewelry industry, where foot presses are used.

The value of proper care and rest during pregnancy is shown by the Leipsic statistics, which indicate that the percentage of premature births was 0.3 and of abortions 2.3 among the volunteer insured, against 1.7 and 15.5 per cent. among the compulsory insured. This difference, Koelsch[24] explains, is due to the fact that the volunteer insured mother pays the cost of insurance and sacrifices more or less of her wage, in order to protect her offspring. The percentage is especially high among workers exposed to lead poisoning: 53.6 per cent. in metal polishers, 22.9 per cent. in type foundry workers, 30 per cent. among the tin-foil capsule workers of Vienna.

Rubner, cited by Koelsch, estimates the number of still-births in certain harmful occupations as between 150 and 170, against an average rate of 33 per 1000 births.

Strassmann's and Falk's statistics show an undue prevalence of menstrual disorders, inflammatory conditions and displacement of the uterus, abortions and diseases of pregnancy among seamstresses using foot-power sewing machines. This excess, amounting to between 40 and 50 per cent., is at-

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tributable to prolonged sedentary and faulty positions, constipation, chlorosis, pelvic congestions, and possibly also active hyperæmia of the pelvic organs caused by overexertion of the lower extremities, since the difference is not so pronounced in hand seamstresses and those using power machines. Similar conditions are observed among laundresses, who moreover not infrequently suffer from varicose veins and ulcers of the leg.

Space will not permit of a more detailed presentation of the statistics so laboriously collected by Koelsch.[24] A few important facts may be summarized as follows: 1. The Austrian and Leipsic statistics indicate that the mortality rates of female workers between the ages of 15 and 60 are distinctively higher than in unoccupied females of the same age period. 2. The Leipsic statistics show that, while the death rate is about equal for both sexes below the age of 15, between the ages of 24 and 29 it is 1.09 per 1000 higher in the female workers, and between 30 and 40 it is 0.70 per 1000 higher than in the male workers. 3. The morbidity rates and duration of illness are likewise considerably higher during the same age period than in male workers. The number of cases of sickness per 100 for female workers was 41.8 with an average duration of 24.6 days, while the rate for male workers was 39.6 and 21.6 respectively. After the completion of the menopause, statistics are in favor of female workers. 4. Diseases of the blood, of the nervous system and of the digestive organs predominate in all comparative statistics. Tuberculosis is less frequent among female workers, 19.2 per 10,000 against 23.2 in the males. During the child-bearing period, however, the rates are excessive, especially during the ages of 30 to 35 years. This difference is less pronounced in the general population and indicates that industrial work combined with maternal functions is harmful.

Accidents and Injuries.—The total number of deaths reported from accidents during the last census year was 57,513, of which 43,414 were males and 14,099 females, and the proportion of deaths from these causes in 1000 deaths from all known causes was 57.6. In 1890 the corresponding proportion was 53.7. In the registration area the rate was 96 per 100,000 of population. In 1890 the rate was 91.9. The rate in the cities was somewhat higher than in the rural districts, and the rate for males was about three times as high (125.4) as among females (42.2). This is due to the more sheltered position of females and because males alone are generally engaged in the more dangerous operations.

The highest death rates from accidents occur in persons 45 years of age and over, and the lowest in children under the age of 15, which indicates that employment in factories, workshops, mines, steel industries, the railway service, etc., influences to a great extent the number of accidents and injuries.

Life insurance and accident policy statistics plainly indicate the danger of occupations which involve contact with machinery. This is generally attributed to individual carelessness or the negligence of others. It would be interesting to know just how much of this so-called carelessness is really

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due to fatigue, which doubtless lowers the power of alertness. Not infrequently accidents from boiler explosions, circular saws, planes, belting and flying fragments are due to a lack of proper safety devices. As might be expected, many of the accidents befall children and inexperienced persons, and take place at night or in badly lighted establishments. The German accident statistics, cited by Roth,[25] clearly indicate that accidents increase with mental and physical fatigue. Upon the assumption that there is one accident for every three working hours during the year, the average number of industrial accidents was as follows:

6– 9 A.M.	1.10	12–3 P.M.	1.02
9–12 A.M.	2.26	3–6 P.M.	2.11
Saturdays, 3–6 P.M.	2.76		

Prof. Imbert at the International Congress on Hygiene at Brussels, 1903, presented similar statistical material. According to Rubner,[26] of 100 accidents 41 befell children under 15 years of age, 36.4 befell persons between 15 and 25 years, 13.1 befell persons between 25 and 40, and 9.5 befell persons between 40 and 60 years of age. The upper extremities were involved in 87 per cent. of the cases, the lower extremities in 7.5 per cent., and the head and trunk in 5.5 per cent.

Swiss statistics show that among 1000 workers accidents occur as follows: Cotton spinners 22.2, millers 28.0, paper industry 31.1, carpenters 35.2, locksmiths 46.9, brewers 66.7, masons 80.5, blacksmiths 93.1, metal workers 102.1, moulders 132.2.

Many of the accidents befall the eye. Magnus attributes 8.5 per cent. of all cases of blindness to accidents. Interesting details will be found under various occupations. The number of accidents in textile mills in this country, considering the large number of fast-running machines, is not large, during a period of almost 5 years at the Pacific Mills, Lawrence, Mass., with about 52,000 employees amounting to 1000. Of these, one was killed outright, one fatally injured, 86 seriously injured (broken limbs or amputations necessary), 910 slightly injured, and two suffered from nervous shock but were physically uninjured. The underlying cause of injury is given as follows: Careless manipulation 539, inattention to surroundings 177, taking chances of being injured—deliberate carelessness, such as cleaning machinery while running, etc., 164, carelessness of fellow-workmen 51, unforeseen liability 60, unclassified 9.

Prevention of Accidents.—Most of the States have taken steps to reduce accidents to a minimum. For this purpose they have enacted laws concerning employers' liability if they fail to provide safety devices for the movable and dangerous parts of machinery. Much has been done also in the prevention of accidents in mines, in the railway service, in steel plants, etc., which will be referred to under specific occupations.

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A careful inspection of steam boilers and examination of engines has materially lessened the dangers from boiler explosions, so that in England there is only about one explosion in 6200 registered boilers. Nearly all states require some form of protection in case of fire, by means of fire escapes, semi-fireproof buildings, and doors swinging outwardly, while a respectable number also provide for automatic sprinklers and other fire-extinguishing methods. For suitable clothing see page 442. For preventive legislation see page 801.

General Welfare Measures for Wage Earners.—Sufficient evidence has been adduced and more will be presented in subsequent pages to indicate that the laboring classes need special protection against the many occupational hazards and injurious environments to which they are exposed. This protection should emanate from the State, the employers, the community and the employees themselves. Each has certain duties and responsibilities, and for success coördination and coöperation are essential. The protection of wage earners should extend to the work and workshop, and in case the employees are housed by the employer also to the living and sleeping quarters, mess rooms, etc.

A sanitary workshop, apart from good management, demands sufficient air-space for each inmate, a suitable temperature, proper ventilation, general cleanliness, sanitary conveniences, separate toilet rooms for men and women, facilities for personal cleanliness, wash rooms, shower baths, dressing-rooms, clothes lockers, lunch rooms, etc., and the State should see to it that the general principles presented in this book are embodied in effective laws. A few States make provisions for "fresh drinking water of good quality" and individual drinking cups or drinking fountains. Some of the States regulate the spitting habit by insisting upon proper cuspidors.

Tenement Factories.—Among the most dangerous forms of workshops is one class which most state laws entirely ignore. For example, under the laws of the State of New York, relating to manufacturing in tenement houses, according to Dr. Anna S. Daniels, 33 distinct industries may be carried on in the living rooms of the workers, because they involve hand-work or simple machinery. There are over 23,000 licensed home factories in the city of New York alone. In addition to wearing apparel for men, women and children, including adornment of woman's dress, the flowers and feathers for her dress, the hats themselves and neck wear of every description, Dr. Daniels found the manufacture of paper boxes, cigars, pocketbooks, jewelry, clocks, watches, wigs, fur garments, paper bags, etc. The articles are not infrequently handled and stored in infected rooms. Of 150 families tabulated by her, 66 continued at work during the entire course of the contagious disease, such as tuberculosis, measles, scarlet fever, diphtheria, etc., for which she was attending the family.

Apart from the occupations referred to, numerous bakeries, candy, ice-cream, milk and butcher shops, bottling establishments, tailor, cobbler and

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other repair shops are carried on in basements under the most insanitary environments.

Construction Camps.—It not infrequently happens that mining, lumber, construction companies, and other large concerns provide board and lodging for their unmarried employees. Again, in a number of the smaller industries, the employees not infrequently board with the family and are obliged to sleep in objectionable rooms.

The baneful effects of overcrowding as observed in the construction camps at Panama have been referred to on page 431. All remedial provisions should come up to a reasonable standard as regards salubrity. Factory sanitation and the matters just alluded to are after all largely questions of public health and should not be left to the discretion of the individual employer, but the principles which ought to be adopted should be embodied in suitable laws and enforced by competent inspectors.

What the Federal Government May Do.—Much excellent work has been and is being done by the United States Bureau of Labor, in the collection and publication of facts concerning every phase of industrial and social betterment. The bulletins issued, if carefully read, cannot fail to exert a tremendous educational influence upon those for whom they are primarily intended, viz., the wage earners and employers. But, while much has been achieved, more remains to be accomplished. It has been suggested that apart from establishing, in connection with the National Museum, a permanent exposition relating to industrial betterment of wage earners, it is clearly the duty of the Federal Government to establish and adopt a standard of industrial hygiene for all the government workshops.

President Roosevelt, in a message to Congress, Dec., 1907, has said "the National Government should be a model employer. It should demand the highest quality of service from each of its employees, and it should care for all of them properly in return. . . . . ."

We regret to say that, with the possible exception of the extraordinary precautions exercised to protect the health of the employees in the operations connected with the construction of the canal on the Isthmus of Panama, the sanitation of offices and workshops for Government employees is not even on a par with some of the best private industrial concerns of this country. There can be no question that model government workshops, and efforts for the promotion of the general welfare of the employees, would prove a salutary precept and example. The General Government is not in a position to legislate for the States, but it can at least enact a model labor and factory law for the District of Columbia and all of the workshops connected with the Army and Navy arsenals—gun factories, powder depots, and clothing depots—and for the immense army employed on the Isthmus of Panama.

Industrial Insurance.—Apart from strictly sanitary measures for the promotion and preservation of health, the Government, as a model employer, should provide some adequate relief in case of sickness, accidents, or dis-

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ability from disease or injuries contracted in the line of duty, and thus initiate a system which has proved to be a vertitable blessing elsewhere. Under the operation of the German law, enacted in 1883, all workmen employed in commerce, industries and the handicrafts, whose income is less than 2000 marks (about $480), must be insured. By special regulation this requirement may be extended to agricultural and household employees. To secure the enrollment of individuals for "sick benefits," the employers, in the industries subject to the law, are required to send to the proper insurance fund the name of each person who enters or leaves their service. The income of the sick fund is partly derived from the dues of members. The amount is fixed by each local association, but cannot exceed 6 per cent. of the member's wages. The employee pays two-thirds of the dues and the employer one-third. The employee's share is deducted from his wages and paid directly to the insurance fund by the employer, when he remits his own share.

A detailed account of the operations of this system will be found in my report as Chairman of the Committee on Social Betterment of the President's Homes Commission, 1908, page 87, from which the following synopsis of practical results is here reproduced.

1. At the end of 1905, in all 70,000,000 pensioners (sick, injured, invalids and their dependents) had received $1,200,000,000 in benefits. The workmen have contributed less than one-half of the premiums and have received $480,000,000, more than they paid out. Property is owned to the amount of $408,000,000, of which $120,000,000 has been invested in workmen's dwellings, hospitals and convalescents' homes, sanitoria, baths and similar institutions of welfare.[27]

2. The financial status of the workingmen has been improved at least to the extent of the benefits received from the amounts contributed by the employers and the Government. Experience has shown that employers have not deducted their share of the dues from wages.

3. The hygienic conditions of the workingmen have been improved, both on account of the safeguards which the accidental insurance organizations require employers to use and because of the special efforts made by the "sick funds" to reduce the sick rate among the members to a minimum. The general knowledge in regard to the preservation and promotion of health, which the "sick fund organization" has disseminated by means of circulars, monographs, popular lectures, etc., has exerted a tremendous educational influence in the promotion of heath and morals.

One of the most beneficient features of the entire system has been that parts of the funds of these organizations are invested in model houses for wage earners, and hospitals and sanitaria for the use of members. It is interesting to note that the Prussian "insurance institute and sick funds" in 1907 alone maintained 28 hospitals and sanitaria, the latter chiefly for consumptives and convalescents. One of the latest features was the estab-

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lishment at Lichtenberg, near Berlin, of a special hospital for sexual diseases in the male, and a sanitarium for nervous and anæmic female wage earners in Pyrmont (Hannover), all upon the principle that it is in the highest degree good economy to restore as speedily as possible the unproductive to the ranks of the producers.

The writer has purposely devoted much space to the German Industrial Insurance system, because he realizes that sickness and funerals are the most potent causes of poverty and distress, and that worry and anxiety often retard convalescence. He knows, from personal knowledge, that prior to 1883 Germany depended upon employers' liability laws, charitable organizations, and private companies for the protection of her wage earners, with very questionable results. While much has been achieved in other directions for the prevention of disease, the most distinct gain in social political endeavors was made by the enactment of these laws, and especially the law of June, 1889, authorizing "insurance institutes" to invest part of their funds in hospitals and sanitaria, thus affording the best possible facilities for speedy recovery, and the prevention as far as practicable of permanent disability.

What the Employer May Do.—Social betterment cannot be dissassociated from industrial betterment, and it is here that the employer can do much for the welfare of his employees. Apart from a cheerful compliance with the laws and ordinances which may, from time to time, be enacted for the protection of the working classes, it is clearly the duty of the employer to promote in every way the efficiency and earning power of the wage earner, and to pay such wages as are necessary to improve the standard of living among poorly paid employees.

In occupations involving exposure to industrial poisons it is a doubtful policy to employ casual labor or to attempt the instruction of new laborers. It has been shown over and over again that accidents and cases of industrial poisoning are less liable to occur in experienced workers. Hence it is far better to take precautionary measures in the first place, and in case of an impending breakdown to consider the question of "change of work instead of change of workers."

There is no doubt that thoughtful employers generally realize that they are not only responsible for the proper technical training of apprentices but also for their habits, and a gratifying number of establishments have made efforts to surround them with opportunities for mental, moral and physical improvement. There is a class of youthful employees, both males and females, for whom the writer begs to enter a special plea; they are entitled to every consideration, because, either as a result of inheritance or faulty environments, they have acquired a general inaptitude; they are perfectly willing to work, but awkward in all their movements—simply do not know how to work—and special pains should be taken to teach them by patient fellow-workmen.

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Industrial Betterment.—Space will not permit to enter into details concerning efforts which have been made by employers at home and abroad in the promotion of the general welfare of the working classes. Every effort in the right direction results in the health and efficiency of employees being promoted, the profits of the manufacturer increased and the quality of the products improved.

Among the most important betterments may be mentioned: (1) the increasing of industrial efficiency, through industrial schools and manual training classes; (2) the care for employees' health and comfort by means of bathing facilities, gymnasiums, calisthenics, base ball, bicycle clubs, dining and lunch rooms, and the furnishing of hot lunches, free or at cost—some establishments even permit dancing during the noon hour—improved sanitary conditions and appliances, lectures and demonstrations, medical supervision; (3) the improvement of domestic conditions by means of improved dwellings, instruction in sewing, cooking and housekeeping, in landscape and kitchen gardening, and in exterior and interior decorations of homes; (4) the care of sick and disabled employees by means of rest rooms, emergency rooms, first-aid outfits, medical attendance and hospital facilities, free insurance, and by the encouragement of beneficial organizations; (5) club organizations for social, recreative and intellectual purposes, by means of free lecturers, libraries, kindergartens and educational classes, social gatherings, summer outings, meeting places, game rooms, banquets, dances, etc.; (6) the encouragement of musical and dramatic clubs and the promotion of spiritual life by means of Sunday schools and general religious work; (7) the cultivation of thrift through savings bank facilities, building associations or provident organizations, rewards for valuable suggestions of employees, for faithful service, or the manifestation of zeal and interest in their employment; (8) the promotion of employees' personal interest in the successful conduct of the business by encouraging and assisting them to purchase shares, financial aid to employees in case of unusual hardships and distress, and the cultivation of cordial and even confidential relations between employer and employee.

What the Public May Do.—It will be conceded that the burdens of improving industrial and social conditions should not be carried by the employers and employees alone. There are many phases of importance, from the standpoint of public health and humanity, which should concern every thoughtful man and woman. Reference has already been made to the dangerous conditions under which many of the trades and occupations are carried on in tenement houses. While this is in part due to the greed of the manufacturer, because it means less factory space, less rent, light, fuel, and a decidedly smaller pay-roll, the consumer is equally to blame because of his constant demand for cheaper goods, quite oblivious to the fact that the garments may be a source of danger from infectious diseases, and are stained with the sweat and blood of helpless women and children.

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During one of the Presidential campaigns a clever orator referred to Glasgow, and told us that 41,000 of the 100,000 laboring families of that manufacturing center lived in one-room tenements, and that this one room for a family of father, mother, daughter and sons told what the wages in Scotland were and how they dragged humanity down into bestiality and misery. It must be confessed that similar conditions obtain in nearly every American industrial city. The effects of such living conditions upon death rates will be presently referred to. In the meantime, it will be readily conceded that the people do not as a rule live in such quarters from choice, but from sheer necessity. Low wages not only compel the working classes to find shelter in houses unfit for human occupation, but also affect their health and the health of their children by insufficient food and clothing; and, last but not least, it means the utilization of child and female labor.

It is to be hoped that public conscience may be sufficiently aroused to insist upon adequate wages for all classes, and that the producer and consumer alike will be willing to assume this responsibility, not as a matter of charity, but in justice to the laboring classes.

Houses for Wage Earners.—In the whole range of social betterment and sanitation, especially in our efforts to combat tuberculosis, no field affords better opportunity for philanthropic work than the erection of sanitary homes for wage earners, at reasonable rentals, the encouragement of cooking schools and the establishment of model lodging and eating houses. The Washington Housing Companies have an investment of over $1,000,000, have paid 5 per cent. annual dividends, from the very inception of the company, and have a surplus fund of over $225,000. London has more than $100,000,000 invested in model tenements.

The housing of the working classes has very properly been made the subject of legislation in many countries, and is a matter in which factory owners, labor unions, and the general public, should be deeply and mutually interested. There are several systems of dwellings for artisans and laborers, viz., individual houses or cottages, rows of houses under one roof, and the so-called "flat." Preference should be given, when practicable, to the cottage system, but in large cities, unfortunately, the value of real estate frequently compels the erection of large tenements. In such an event the State should insist upon hygienic requirements as regards air-space, light and ventilation. No home can be considered sanitary where one room has to answer the purposes of living room, sleeping room and kitchen, or where the water-closet or privy is used by more than one family.

With the present rapid transit facilities, in every city, our voice should be clearly in favor of individual homes; and when this is impracticable, we should insist upon broad streets and deep yards. No more than 68 per cent. of the lot should be covered by the house, and the height of the building should never exceed the width of the street. The baneful effects of tenement houses should be avoided, as infectious diseases are more liable to

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spread, in consequence of aerial infection and a more intimate contact of the occupants.

Apart from structural defects, there is no doubt that the death rate is largely determined by the number of occupants to a room. Russell has shown that in Glasgow, when the average number of persons to each room was only 1.31, the mortality was 21.7 per 1000, and when the number of occupants amounted to 2.05 for each room, the mortality reached 28.6 per 1000.

The death rate at Berlin, in 1885, among the 73,000 one-room tenements was 16.35 per 1000, against 5.4 per 1000 among 308,000 residents occupying four or more room apartments.[28] Neumann,[29] in investigating 2711 infantile deaths in Berlin, found that 1792 occurred in one-room apartments, 754 in two-room apartments, 122 in three-room apartments, and 43 in apartments of four rooms and over.

Insanitary dwellings are to be found everywhere, particularly in older cities erected at a time when the principles of sanitation were comparatively unknown. One of the most important municipal problems is to correct existing evils by the enactment and enforcement of suitable laws. It requires, however, a strong public sentiment to bring about a complete and satisfactory reformation, as evidenced by the housing movement everywhere.

The housing conditions of the least resourceful people have been, and are even now, more potent than any other factor in helping to swell the frightful mortality from consumption and other so-called house diseases engendered by unwholesome environments. The existence of disease-breeding habitations is a reflection upon Christian civilization, and there should be sufficient human sympathy to provide decent, healthful homes for our wage earners, who constitute, after all, the bone and sinew of the country. This is one of the occasions when we may well act as our brother's keeper.

The history of improved dwellings reveals everywhere a lessened death rate, and the experience of the Washington Sanitary Improvement Company is equally gratifying. During the year ending December 31, 1906, the apartments were occupied by 778 adults and 380 children, total 1158; births, 39, and only 16 deaths, 10 adults and 6 infants; a death rate of about 13.7 per 1000, which, with all due allowance for the average age of the occupants, shows a remarkably low mortality, when compared with the general death rate among the white population of 15.16 per 1000.

The regeneration of the housing conditions for the least resourceful people is the great sanitary and social problem of the twentieth century. Take away the hovels and filthy places, let sunshine and pure air circulate through the homes, and teach them habits of cleanliness and responsibility, and the first step toward the elevation of the degraded and the education of the ignorant will be taken, not only in the warfare against tuberculosis and other diseases engendered by insanitary surroundings, but also in the battle for a higher moral and social standard.

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Lodging Houses or Homes for Wage Earners.—Those who have read "The Long Day" cannot fail to be impressed with the just criticism of our present system of homes for working girls. The author makes a strong plea for homes designed after the Mills Hotels for working men; no charity, but so built and conducted that they will pay a 4 per cent. rate of interest upon the money invested. "A clean room and three wholesomely cooked meals a day can be furnished to working girls, at a price such as would make it possible for them to live honestly on the small wages of the factory or store." It is to be hoped that the simple but truthful story of the working girl will be read and her appeal for industrial and social betterment answered. To supply the need spoken of, together with the establishment of cooking schools, and kindergartens, so that the children of toil may at least have an opportunity to learn to work intelligently, may be regarded as a suitable field for practical christianity, and would do much toward narrowing the breach which now exists between the church and wage earners, and between capital and labor.

While the character and variety of the food now served in boarding houses is very much better than it was years ago, it is not what it should be. The chief fault consists in improper cooking, and the widespread error of consuming a cold meal from the lunch or dinner bucket.

The art of cooking, and how to supply good wholesome food in proper quantities, should be made the subject of popular instruction. The "Ladies Sanitary Association of England," deserves credit for having taken this matter in hand, especially since experience teaches that nothing prevents the abuse of alcohol so much as a sufficient and palatable supply of food.

The establishment of public kitchens and eating houses for unmarried laborers, conducted upon practical sanitary and economic principles, would prove a great blessing.

What the Employee May Do.—It must be conceded that all methods proposed for the alleviation of the effects of adverse working conditions are prompted by the spirit of humanity and as a social duty. Hence it is reasonable to expect that wage earners should in every way coöperate and avail themselves of the various safety devices and preventive methods and not underrate their importance in the protection of health, life and limb.

While it is criminal for employers not to provide suitable protection it is equally culpable on the part of the operatives to disregard such preventive measures. So, for example, it is not a pleasing reflection when we are told by the Medical Inspectors of the State Board of Health of Massachusetts, that a considerable number of employees remove the hoods over emery wheels on the ground that they interfere with their work, and that they insist iron enriches the blood. Professor Harrington in speaking of respirators says, "Aside from the discomfort caused, the operatives have another and a senseless objection to their use, women complaining that they are made to look ridiculous, and men being moved to discard them by the jibes of their

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more reckless fellows." Dr. Farrand, former Secretary of the National Association for the Study and Prevention of Tuberculosis, spoke to me of the great difficulties he and others had encountered, in New York and New Jersey, in inducing the operatives to give safety devices a fair trial. My personal experience and that of other social workers has at times been equally discouraging. Time and again have the good intentions of employers been frustrated by "self-willed" workmen, who professed to know better. Even in Government arsenals I have found men working in high explosives without rubber gloves and respirators, although provided by the Government.

With time and patient education these difficulties are surmountable, and it is just here that the intelligent worker, the man who appreciates the value of his health and his duties to his family, will prove an efficient coworker in the prevention of misery and distress. It is hardly fair to characterize certain trades as dangerous, when experience has shown that no harm results when proper safeguards have been taken. In the consideration of this question the personal element of the workmen, their habits, mode of life, food, home environments, etc., cannot be ignored.

There are a number of occupations in which the alcohol habit prevails to an unusual extent, perhaps because of the character of the work, perhaps as a result of association, and it would not be fair to attribute the ill health of the operatives altogether to the character of the employment. Again, many persons are engaged in occupations for which they are not physically fitted, while others ruin their health by vice, dissipation, improper food, and insanitary environments at home. The writer is fully convinced that the home, and home influence, play a very important rôle in the causation of so-called "occupational diseases." As early as 1898, under the leadership of Surgeon General Sternberg, he helped to establish a company for the erection of sanitary homes for wage earners, at reasonable rentals. Later in 1908 as Chairman of the Committee on Social Betterment he discussed in detail the numerous contributory factors just alluded to.

Hygiene of the Workmen.—*House and Home.*—Special pains should be taken in the selection of living quarters no matter how humble they may be. With the excellent motor facilities, there is no reason why crowded tenements should be chosen and preference should always be given to individual homes, or apartments, in not exceeding two-story tenements. The Germans have an old but true proverb—"Where the sun does not enter the doctor surely will;" hence dark gloomy and damp houses should be avoided; mouldy spots on the walls or ceiling and a close musty odor indicate dampness, and cheap rents should prove no inducement to occupy such quarters. Leaky roofs and down spouts, or a pile of ashes against a brick wall, may keep the house damp, and the causes should be promptly removed. In all such instances, as well as in the occupancy of a recently constructed house, it is very desirable to dry out the house by heat and open windows.

Since we know that the mortality from contagious diseases increases in

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proportion to the number of inmates of the rooms, hygiene requires that even the most modest dwellings should afford sufficient room to prevent overcrowding. Ventilation is always necessary, but open windows are especially indicated at night, as nothing can take the place of pure fresh air in small quarters. This may be effectively accomplished without the danger of draughts by opening the windows in the bed room from the top, and those of the adjoining room at the bottom.

There are many families who properly insist upon having a sitting room or parlor, which is most commendable if the bed rooms are large enough to afford at least 500 ft. of cubic air-space for each occupant. If they do not it is desirable, after proper airing of the larger rooms, to utilize them for sleeping purposes, for it must be remembered that the air of habitations is vitiated by the consumption of oxygen and the exhalation of carbonic acid. The airing of rooms is even more essential in cold weather, because of the additional pollution by carbonic acid from lights and fires. There are families, unfortunately, who for various reasons are obliged to live, cook and sleep in one room, and for whom the question of fresh air is therefore of vital importance. Such families should not hesitate to avail themselves of the benefit of fresh air, especially when medical science has demonstrated the advantages of fresh and even cold air in the treatment of consumption and pneumonia, provided the body is kept warm by sufficient bedclothes. Night air, contrary to popular opinion, is not unwholesome. The only danger is from mosquitoes, which should be excluded by proper screening of windows and doors.

Care of the Skin.—The skin is supplied with a network of blood-vessels and nerves, and is a sensory, respiratory, excretory and heat-regulating organ. As a sensory organ, it combines with the tactile functions the power of perceiving impressions of warmth and cold. The respiratory functions of the skin are limited, to be sure; nevertheless, small quantities of oxygen are absorbed and carbonic acid is eliminated. Apart from this, the skin of an average adult eliminates, through the sweat glands, about 2½ lb. of water a day. Human sweat contains about 2 per cent. of solid constituents, mostly in the form of waste matter or impurities, and the odor varies in different regions of the body and in different races. The skin also secretes a fatty substance through the sebaceous glands. As the water from the skin evaporates, the solid matter remains upon the surface, combines with dirt, harbors germs, and readily undergoes decomposition. This; apart from the disagreeable odors so characteristic of unclean persons, also tends to macerate the skin and is liable to produce "galling or chaffing," pimples and boils. The accumulation of this matter also tends to close the pores of the perspiratory and sebaceous glands and to throw the work of eliminating the impurities upon other organs.

A normal cutaneous function is doubtless of great hygienic importance, as shown by the occurrence of many diseases following its suppression, be-

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cause in such an event, in addition to the retention of the waste matter in the blood, work is thrown upon the kidneys and other eliminating organs, and these, if already weakened, naturally break down. Since the functions of the skin depend not only upon its anatomical intactness but also upon cleanliness and a proper tone of the cutaneous vessels and nerves, a rational care of the skin demands: (1) That it should be freed regularly from the secretory products and particles of dirt; (2) that the cutaneous nerves retain their normal excitability or, when impaired, that they regain their tone; (3) that we assist the skin in its heat-regulating functions, so that it may not be overtaxed—all of which may be accomplished by ablutions, baths, and suitable clothing.

Ablutions and *Baths*.—Regular and systematic ablutions with soap and water are requisite for reasons already given, and are especially necessary for persons engaged in dirty work or exposed to poisonous dust. In addition to the use of soap, vigorous friction with a brush should be employed, not omitting the finger nails, on account of disease germs and industrial poisons. The hands should always be washed before meals and after going to the toilet.

The water for general baths should not be too hot, as this would relax the skin and increase the susceptibility to catching cold. If the bathing is not done in a bath tub, it will be well to wash and dry part of the body at a time. In any event the surface should be wiped dry and hard, especially the hair, since wet hair is calculated to produce colds. It is always a good plan to wash the neck and chest with cold water, so as to harden the skin.

The cold bath is usually taken in a tub or by means of a shower or needle bath, at a temperature of about 65° for adults. It should not last over 3 minutes. Cool baths vary from 65° to 80°. Tepid baths are taken at a temperature of between 80° and 90°, continued from 10 to 15 minutes. Warm tub or shower baths vary from 90° to 100°, and are generally employed for their cleansing effect. In addition, there are steam or Russian baths, Turkish or dry hot-air baths, river and ocean baths, swimming pools connected with public baths, medicated baths, etc. Swimming baths are particularly useful, as they also afford an opportunity for muscular exercise, and, as the temperature of the water is rarely above 80°, such baths are both cleansing and stimulating, and therefore an excellent tonic for the skin. All baths should be followed by a cool douche, and friction with a rough towel should be employed until the skin is in a general glow. The value of bathing is so fully appreciated that the building regulations of Washington City compel a bath room for every apartment offered for rent. No community should fail to make provisions for public baths for summer and winter. The necessity for washing and bathing facilities in connection with industrial plants has been pointed out on page 443.

Clothing.—The object of clothing, apart from the moral and æsthetic aspect, is to aid the skin in its various functions. It should, therefore, afford protection against heat and cold, as well as rain and mechanical irrita-

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tion. Clothing must be adapted to climate and season, and extremes should be avoided. As a general rule warm woolen goods are best suited for winter wear, and cotton or linen for warm weather. It should be understood, however, that flannels absorb more dirt, odors, germs and water than linen or silk, while cotton occupies an intermediate position. The question of wet clothing, whether from perspiration or rain, is important, as the drying of clothing on the body involves an expenditure of animal heat, and it is not a matter of indifference whether this takes place rapidly or slowly. It is a fact that a wet cotton shirt or sweater feels more uncomfortable and colder than a wet woolen garment, because the cotton garment dries more rapidly and abstracts during the same time more animal heat than flannels. This is not without a practical bearing, as it teaches that persons who perspire easily will do well to wear flannels next to the skin. This is all the more important when they are liable to drafts or abrupt changes in temperature.

As a protection against cold, wool is superior to either cotton or linen, and should be worn for all underclothing. In case of extreme cold, besides wool, leather, fur or waterproof clothing, on account of their impermeability to air, are useful. As a protection against cold winds, for equal thickness leather and India rubber take the first rank, wool the second. As a protection against rain, India rubber or oiled canvas clothing is the best, but it is an exceedingly hot dress, owing to its impermeability to air, which causes condensation and retention of the perspiration. To overcome this objection, Dumas suggests a material which is waterproof and yet permeable, prepared as follows: The garment is placed in a 7 per cent. solution of gelatine, heated to a temperature of 100°F. After immersion for a few minutes it is dried in the air and after drying it is placed in a three-fourths of 1 per cent. solution of alum and again dried.

As a protection against heat in the shade, the thickness and conducting power of the material are the only factors to be considered. Texture has nothing to do with protection from the direct solar rays; it depends entirely on color, and white is the best. As a protection against fire, leather clothing is generally worn. The fabric can be rendered non-inflammable by the addition of 20 per cent. of tungstate of soda and 3 per cent. of phosphate of soda to ordinary starch sizing. Cotton or linen goods may be treated simply with starch and borax, in the proportion of a teaspoonful of borax to ½ pint of starch. See also special work suits, page 442.

Clothing as a Cause of Disease.—Clothing may impair the functions of the body and cause disease: 1. By improper fitting, which leads to compression of blood-vessels and nerves and interferes with the normal position of organs and the movements of the body. 2. By improper selection of material, affording either insufficient protection or overheating a part or the whole of the body; improper material may also produce irritation or interfere with the ventilation of the skin. 3. By wet clothing which, in drying, may abstract sufficient animal heat to cause peripheral irritation and reflex internal con-

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gestions. 4. By poisonous dyes, such as compounds of arsenic and antimony, chrome yellow, zinc chloride, and some of the aniline colors. The toxic symptoms may manifest themselves by general impairment of health or by local infections of the skin. 5. Clothing may harbor disease germs, and a number of instances are on record in which itch, smallpox, tuberculosis and scarlet fever have been spread by second-hand clothing and bedding. This points to the necessity of thorough disinfection.

The Head Dress.—As long as the head is covered with hair, the head dress should be permeable and not too warm, thus less headache may be induced; on the other hand, insufficient covering may produce neuralgia and rheumatic affections.

The head and eyes should always be protected from the direct rays of the sun, and for this purpose broad-brim, dark felt hats or caps for cold weather and straw or some other light-colored material for summer use are the best.

For the Neck.—Nothing should be worn around the neck which would overheat the parts, dilate the blood-vessels, and render the skin sensitive. The collar should be loose fitting, so as not to compress the blood-vessels. The neck ought to be bared as much as possible, and hardened by frequent ablutions with cold water.

For the Body.—For under garments the union suits are the best. They should secure a normal amount of warmth and be so arranged as not to interfere with the free movement of the chest, or compress or displace the abdominal and pelvic organs. For these reasons corsets and waist bands are wholly inadmissable. Suspenders should be worn by both sexes, or women may wear a bodice arranged for the attachment of skirts, so as to suspend their weight from the shoulders. Steel corset stays and tight lacing cannot be too strongly condemned, because there is ample evidence that they have caused displacement and disease of the abdominal and pelvic organs. The trousers must be sufficiently loose around the waist and elsewhere to permit of free circulation of blood.

Foot Wear.—The stockings should be made of some warm, permeable, material such as cotton, merino or wool. Tight elastic bands or constricting garters are liable to produce varicose veins. Holes and rough darning of socks should be avoided. Boots and shoes are intended to protect the feet from the uneven and rough surfaces of the ground, from cold, wet and even heat, and must be constructed so as to meet these requirements. It is needless to insist that they should be patterned after the foot. The sole of a shoe should be so constructed that the great toe touches it in such a way that a line projected posteriorly through the middle of this toe will strike the middle of the heel. The heel should be broad and of medium height, so as not to throw the weight on the toes. Across the tread and toes the sole should be sufficiently broad to permit of lateral expansion. The uppers should be soft and flexible, but not too roomy, and should fit snugly around

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the ankles and insteps. In occupations involving exposure to wet, the feet should be protected by rubbers or preferably wooden shoes.

Since cleanliness, in body and clothing, is next to Godliness, frequent bathing and change of underwear are desirable. It is also a good plan to use nightshirts or pajamas, so as to afford an opportunity for a thorough airing of the underwear worn during the day.

Care of the Ears and Nose.—These organs should be kept clean. There is danger from lodgment of foreign bodies, which may impair the hearing for life, especially when injudicious attempts at removal have been made. In such instances it is always best to consult a physician. The accumulation of dust in the ear may give rise to inflammatory conditions and favor the development of impacted ear wax.

The chief function of the nose evidently is to arrest more or less of the dust and germs upon its mucous surfaces, and in cold weather to warm the inspired air. Much of the dust lodged in the nasal cavities will sooner or later find its way into the stomach, and if it contains toxic substances, may cause industrial poisoning. In cement workers actual concretions are formed in the nasal cavity, hence the importance of cleanliness and of breathing through the nose instead of the mouth. In order to do this without discomfort, it is necessary that there be no obstruction in the nasal passages, and persons suffering from catarrh or other symptoms of difficult breathing will do well to undergo treatment. A weak solution of boric acid or cooking salt (a teaspoonful to a teacupful of warm water), gently snuffed from the palm of the hand or used by means of an atomizer, is very useful in the prevention of troublesome and even serious affections of the nose.

Care of the Mouth and Teeth.—It is important that the mouth and teeth be kept scrupulously clean at all times, but especially in occupations involving exposure to toxic dust and corrosive fumes, such as phosphorus, mercury, etc. In addition to the use of the washes just mentioned, with which the mouth and throat may be rinsed, the teeth should be well brushed before meals, in order to remove any industrial poison which may have lodged. Quite apart from this danger, oral hygiene demands that the teeth be brushed upon rising, after each meal, and before retiring, and the gums rubbed with a soft cloth saturated with a strong salt solution. Apart from the corrosive action of fumes and industrial poisons, the temperature of food and drink plays an important rôle in premature decay of the teeth. It has been shown that a sudden change in the mouth from hot to cold causes the enamel to crack, and leads to decay, because the microbes, ever present in the mouth, find these fissures a suitable lurking place for their destructive work. Food lodged between the teeth naturally offers a suitable medium for the multiplication of these germs. Hence the necessity of cleanliness of the teeth, which is best secured by brushing them with water and castile soap. In case particles of food cannot be thus dislodged, a soft silk thread drawn between the teeth will accomplish the purpose more effectively and wisely

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than a toothpick. The temperature of food and drink, in order to avoid cracking of the enamel, should not exceed 120°F. or fall below 40°F. This question also plays an important rôle in the causation of dyspepsia.

If the teeth or gums are already painful or show evidence of decay, accumulation of tartar, or the presence of pus, a dentist should be consulted. Many cases of obscure nerve lesions and septic infections have been traced to unsuspected pus cavities in the mouth. It is a wise plan to have the teeth examined once every 6 months, and, as there are free dental infirmaries, the question of cost need not deter even the least resourceful people.

Care of the Eyes.—The protective measures in the way of suitable eye shields for the prevention of accidents which may befall the eyes have been fully set forth on pages 307–316. Whenever there is any evidence of redness, pain, inflammation, watering or "mattering" of the eyes, a physician should be consulted. While free bathing with cold water or a weak solution of boric acid may exert a tonic effect, especially in dusty occupations, the value of good eyesight is too great to trust to domestic remedies or even to the services of an optician. Whenever there is any difficulty in reading, accompanied by pain or headache, an oculist should be consulted.

Care of the Hair.—From what has been stated in connection with industrial poisons, it is apparent that the head, hair, and beard offer a suitable place for lodgment of dust and condensation of toxic vapors. Apart from the local irritant effect, there is a certain amount of danger from poisoning by absorption and by ingestion of particles lodged in the beard.

It is well, therefore, for workers in all dusty occupations, especially those exposed to industrial poisons, not to wear beards of any kind, and to keep their hair well trimmed. In addition to this precaution suitable work caps of some light impermeable material, like oiled muslin, should be worn, and the hair thoroughly washed upon cessation of the work.

Care of the Hands and Nails.—The finger nails should be kept short and hang nails properly cared for, so as to prevent absorption of industrial poisons and septic germs. In addition to frequent ablutions, there is need in many occupations for the use of rubber or leather gloves. This is especially true of occupations involving exposure to strong acid or alkaline solutions, and numerous industrial poisons, especially in the coal-tar and dinitro-compound industry. The application of cocoa butter, vaseline or cold cream is useful when gloves are impracticable, in certain occupations.

Care of the Feet.—Proper foot wear will prevent corns and bunions, which are often a source of great suffering. It is important that the foot wear be intact when exposed to industrial poison, such as lead, chrome, and dinitro-compounds, etc., in order to prevent absorption. Warm foot baths and change of stockings are desirable upon cessation of work, not only in dangerous occupations but also when the feet ache from prolonged standing and profuse sweating. The application of a weak solution of alum or chlorinated

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soda is useful in case of excessive and offensive perspiration of the feet. (See also page 445.)

Care of the Bowels.—All sedentary occupations predispose to constipation, which in turn favors pelvic congestions, hemorrhoids and uterine disorders, not to mention the equally important consequences of auto-intoxication, which is responsible for the anæmia and disorders of the nervous system so common in persons with constipated habits. In some instances this condition is due to neglect to answer the call of nature. Regularity of the bowels, at least one evacuation a day, is essential for health, especially in workers exposed to lead and arsenic. The most natural remedy to correct constipation will be persistent effort to secure evacuations every day at a fixed hour, aided by the use of succulent vegetables, such as cabbage, spinach, greens, onions, rhubarb, "sour kraut," fruits, like stewed prunes, figs, apples, etc., and from five to six glasses of water a day. Graham or rye bread, oat meal, wheaten grits, butter or butterine, olive oil, are also useful. Milk, cheese, eggs, rice and tea are constipating.

Exercise, which means muscular activity, yields heat and energy and involves a more rapid combustion as shown by the increased elimination of carbon dioxide. Exercise leads to increased muscular development, while inactivity causes a wasting or fatty degeneration of the muscles.

The influence of exercise upon the *pulmonary functions* is well marked since, owing to increased oxidation, a larger amount of air is required in consequence of which the number of respirations is increased. Moderate exercise therefore tends to produce better oxygenation of the blood, increased lung capacity and copious ventilation of the air cells. On the other hand, if the exercise is violent or done at a rapid rate, the respirations become shallow and sighing and thereby materially interfere with the circulation in the pulmonary capillaries and the proper oxygenation of the blood.

The effects of exercise upon the *circulatory apparatus* are also quite marked, as shown by increased force and frequency of the heart's action. During ordinary exercise the action, although accelerated, is uniform and regular, but during violent or long-continued exercise the muscles of the heart like other muscles become tired, as shown by a dicrotic and intermittent pulse. Palpitation of the heart, a rapid irregular pulse and inequality of its volume are indications for rest and of danger from the continuance of the exercise.

Exercise increases the *cutaneous functions*, because the skin in consequence of the accelerated circulation and heat production receives more blood, and in order that the animal temperature may be kept normal copious perspiration takes place.

Owing to the increased elimination of water by the skin, the amount of urine is usually lessened, but the solid constituents, especially the inorganic salts, are increased. The amount of urea is not increased and may even be diminished, in which case, however, there will be a compensatory increase during the interval of rest.

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Exercise exerts a decided influence on the *digestive functions*. In order to compensate for the increased consumption of carbon and water, there is a desire for fatty food and liquids, the digestive functions are also stimulated, but exercise immediately before or after meals retards digestion, because the muscles calling the blood from the stomach and internal organs, prevent the proper formation of the digestive fluids.

There is no doubt that the *brain and general nervous system* participates in the benefits of improved nutrition, secured by proper physical exercise. This is well illustrated in the person of Mr. Roosevelt whose mental and bodily vigor are of the highest order, although as a youth he had a delicate physique.

Every one is familiar with the fact, that trained muscles respond more promptly to volition, a matter of importance in many pursuits in life.

Effects of Overexercise.—If a certain muscle or group of muscles undergo severe and protracted exercise, they become tired and it requires considerable stimulus and will power to set them again in action. If persisted in, the muscular contractions become less uniform, trembling and even muscular spasms may ensue, followed by relaxation and soreness of the parts. These phenomena disappear upon rest, but increase in intensity and lead to complete inefficiency upon continuance of the exercise or work. The tired or exhausted feeling of the muscles is believed to be due to an accumulation of the waste products of their activity (fatigue toxins) and after these products are eliminated or neutralized the muscles resume their function. This, however, is probably not the only cause, for we know that a certain amount of deprivation of oxygen, or rather of arterial blood, diminishes muscular energy and leads to muscular exhaustion; hence rest is essential in order to take in a fresh store of oxygen. The elimination of waste products is important as their retention renders the blood impure and predisposes to disease. In addition it should be remembered that the heart and lungs during hard work or exercise are severely taxed, and that the wear and tear from increased blood pressure and accelerated respiration involves the expenditure of considerable nervous energy, which adds to the tired and exhausted feeling. Da Costa has described a weak or irritable heart especially common among soldiers and pedestrians which he attributes to overexertion and cardiac strain. Aneurysm and rupture of blood-vessels and of the heart, though uncommon before middle life, have followed overexertion and hypertrophy and dilatation of the cardiac muscles are not infrequent results. A person with valvular disease should avoid violent exercise by all means.

General Rules and Kinds of Exercise.—From what has been said it is apparently not a matter of indifference as to how we employ our muscles. Insufficient exercise not only affects the size and energy of the muscles, but also impairs the respiratory, circulatory, cutaneous, digestive and intellectual functions, and even the quality of the blood itself. The exercise of only certain groups of muscles cannot produce a harmonious or symmetrical develop-

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ment of the body and may cause disease as shown by the numerous deformities and neuroses in certain occupations. The subject of exercise is of less importance to wage earners, whose work requires fairly vigorous exercise in the open air; indeed there are many, who are already overworked, but to the men and women, including brain workers, who lead a sedentary indoor life, with little or no muscular exertion, the question of exercise is of the utmost importance. Among the many excellent means of muscular exercise are walking to and from the work. Professor Haughton has shown that walking on a level surface at the rate of 3 miles an hour is equivalent to raising one-twentieth part of the weight of the body through the distance walked. The work done by a person weighing 150 lb. in walking a distance of 3 miles would be equivalent to lifting 52 tons 1 ft. high. Most authors believe with Parkes, that a healthy man ought if possible to take a daily amount of exercise equivalent to lifting 150 tons 1 ft. high, which would be equal to a walk of about 9 miles, but as there is much exertion even in ordinary business this amount may be greatly reduced.

Swimming calls into play all the voluntary muscles, tends to expand the chest and causes extension of the vertebral column and is therefore especially suited to correct the effects of sedentary habits and as the beneficial effects also extend to the skin, this exercise cannot be too strongly recommended for both sexes. *Dancing and skating* also tend to expand the chest and develop the muscles of the extremities, and are beneficial provided the exercise is carried on in a pure atmosphere and is neither too violent or protracted. *Rowing* develops to a marked degree the muscles of the arms, chest and back, and increases the vital capacity of the chest, but in boat-racing the heart and lungs are overtaxed. *Golf* is especially suitable for persons who lead a sedentary or indoor life and can be safely recommended to all, where age precludes violent exercise; the body is gently exercised in all its parts, and there is no danger of overtaxing any particular organ. *Tennis*, *football*, *baseball* and other out-door sports are more violent forms of exercise; they bring into play nearly every muscle in the body and are valuable, but if pushed too far, the heart and lungs, especially in football, are severely taxed. Among other forms of exercise should be mentioned bicycle and motor riding, clubs and dumb bells, swinging on the trapeze, hanging by the arms and legs, bowling, military drills and many of the regular gymnastic exercises. An important point is to vary the exercise in order to call different groups of muscles into play and thus produce a symmetrical development of the body. Even Celsus advised to swim, ride, fence, sail, row, shoot and fish. All these sports will develop the muscles, increase the chest capacity, stimulate nutrition and the action of waste eliminating organs. But it should be remembered that the greatest good is accomplished by moderate and gradual exercise and that violent exercise like all extreme measures is hurtful. As to the amount of exercise perhaps as good a rule as any is laid down by Lynch in his "Guide to Health" that the lean should exercise to the glow point, or until their bodies

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and spirits are heated for that will fatten them, and the fat should exercise to the sweating point. He considers hypochrondriasis and hysteria the special punishments of ease, affluence and indolence and that the more luxuriously a man lives the more exercise he needs.

When in the course of exercise the body becomes fatigued or the heart and respiration embarrassed, rest is indicated. Exercise should not be undertaken, when the circulation is already excited, or while important organs of the body are performing their functions, as for instance, immediately before or after meals, or during the menstrual period. It would also be obviously wrong to begin with violent exercise, or to allow sudden cooling of the body. Attention should also be paid to proper clothing, which should not be too warm or tight fitting, as such garments result in accumulation of animal heat and interfere with free movements of the heart, lungs and muscles. After active exercise the body should be washed with soap and water to remove the secretions from the sweat and sebaccous glands and the surface should be well dried. If a bath is impracticable and the body is in a state of perspiration, a woolen sweater is the most appropriate garment to prevent chilling of the body.

Brain Work.—Our knowledge of the effects of brain work on metabolism is not at all satisfactory. Hammond, Gamgee and Paton determined an increase of urea in the urine, during and after active mental labor, but Caceneuve and Speck were unable to confirm these observations. A similar difference of opinion exists as to the amount of phosphoric acid elimination. Immermann observed an increased elimination of CO_2 during brain work, which is confirmed by Speck, but he attributes the excess not to the mental efforts, but to the attending muscular movements.

Some authors claim to have observed an increased animal temperature, during severe mental labor, but even this is attributed to the accompanying muscular activity. In the light of this conflicting testimony it is evident that the influence of brain work on metabolism requires further elucidation. Practical experience teaches, however, that studious persons, however well fed, are rarely corpulent, and this at least points to an increased consumption of carbon. After prolonged mental effort, symptoms of mental fatigue, such as weariness, headache, inability to think clearly or to concentrate our ideas, amounting at times to complete apathy and disgust may ensue; these symptoms disappear upon proper rest, but unless sufficient recreation is taken, this condition is liable to increase the intensity of the headache, and produce irritability of temper, restless sleep, chasing of ideas, impaired memory, and even symptoms of mental exaltation or depression, and other typical symptoms of neurasthenia. The reason for this is, that every mental effort involves an increased flow of blood to the brain, and unless followed by intervals of rest, this physiological hyperæmia may become permanent, interfere with the nutrition of the working parts of the brain, and thus lead to pathological changes in the brain cells. It is therefore essential not to overtax the brain,

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and to allow a few minutes for the restoration of the intracranial circulation, after every continuous mental effort of 50 to 60 minutes.

While it is true, that the mind may be stimulated to perform an immense amount of labor, it is evident that this involves much danger, and neither reason nor common sense can justify the employment of such stimulants.

There is no doubt that great mental work can be borne without serious effects if hygienic principles of diet, exercise, recreation, and other diversions are attended to, not omitting the elements of hope and cheerfulness, which are great aids, while monotony, worry and anxiety have a most depressing effect and probably have produced more neurasthenics than hard mental work.

Sleep.—Rest and sleep are Nature's remedy to overcome the effects of mental and physical fatigue. We are still in ignorance of the exact nature of sleep, or the suspension of the automatic activity of the brain. Some authors maintain that it is caused by an accumulation of waste products (fatigue toxins) in the blood and central nervous system, and that it ceases with the elimination of these products. Others believe that sleep results from the exhaustion of the supply of intramolecular oxygen, while still others attribute it to a temporary anæmia of the brain. On the whole, there is much reason for assuming that the refreshing effects of sleep are due to the elimination of waste products from the system and the absorption of a fresh store of oxygen. Sleep in an abundance of fresh air is always more restful. Adults require from 7½ to 8 hours. If the amount is materially lessened, languor, pallor and nervous irritability may be observed. Excessive sleep is harmful, as it tends to produce sluggishness of the bodily functions, lessens tissue metamorphosis and favors the deposition of fat. Irregular bed hours are not calculated to promote a sound and refreshing sleep, as the human body resents irregular habits of all kinds. The objections to all avoidable night work are largely based upon the fact that normal sleep is seriously interfered with in day time by light, noises and in hot weather also by heat.

Bed and Bedding.—Since about one-third of our life is spent in bed, something should be said of this article of comfort and necessity. As the object of the bed is to promote a refreshing sleep, it should be long and broad enough to permit of the necessary extension of the body; it should be elastic, so as not to compress the soft parts unnecessarily, and it should be warm, but not too warm. Metallic bed steads are preferable to wood, because less liable to be infected with insects; they should be provided with a woven wire mattress which admits of free circulation of air. Upon this may be placed a mattress of hair, felt, cotton or excelsior. Pillows should be preferably made of horsehair. Feather pillows are too heating for the head, unless a layer of paper has been interposed. High pillows are objectionable, as the position of the sleeper would impede the movement of the diaphragm. Sheets and pillow cases of cotton for winter and of linen for summer are

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necessary to prevent irritation of the skin and soiling of the mattress, pillows and blankets.

The most suitable coverings for a bed are woolen blankets; they are warm and their permeability admits the escape of gases. For warm weather a cotton quilt or comforter or even a linen sheet is preferable. The bedding should be aired every morning and exposed, whenever practicable, to sunlight, which is nature's purifier and destroys all forms of germs.

"Featherbeds" and "down quilts" are warmer than blankets, as the air contained in the feathers is a bad conductor of heat, but they are suitable only in very cold climates, or for anæmic and delicate individuals, because they overheat the body, cause dilatation of the cutaneous vessels and consequently relax and impair the tone of the skin.

The sleeper should lie with his head slightly raised, preferably with the body inclined to the right side. He should rise rather slowly from the recumbent position, since a sudden change to the erect position, not only accelerates the heart's action, but also changes the blood distribution too abruptly. The bed should be so placed that the occupant is not disturbed by the influence of light. A sound, refreshing sleep can be had only when the senses are no longer stimulated, and light is a stimulus which acts even through the closed eyelids.

Alimentation and Food.—The human body needs food, air and water for its growth and maintenance and the fact that proper nutrition of the body is important for efficiency, and the enjoyment of health, has long since been recognized.

The body is composed of about 60 per cent. of water, 19 per cent. of protein compounds, 15 per cent. of fats and 6 per cent. of mineral salts, all of which are sooner or later consumed, and must be replaced if health and life are to be preserved. The chief objects of food are to form the material of the body and to repair its waste; also to yield heat, to keep the body warm, and muscular and other power for the work it has to do. An engine cannot run a machine or draw a train without fuel, which is converted into energy or power. In the same way the body must have fuel in order to generate the power expended in the action of the heart, lungs and ordinary muscular movements, and the various activities of man—whether it be a mason in laying stone or brick, a carpenter in sawing or driving nails, a woman who sweeps or does her household work, children who romp and play, or in any of the avocations of life. (Langworthy.)

Food stuffs are classified according to their proximate composition as follows: 1. *Organic*, which includes (*a*) *nitrogenous food* material commonly called protein, such as the lean part of meat, milk curds, dairy products, eggs, the gluten of wheat and other cereals, the legumin of peas, beans, lintels, peanuts, etc. The proteids of the human body are made up of the same chemical constituents and, hence, the proteids are the chief tissue formers of the body. But apart from this purely plastic function, they can

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also take the place of fats and carbohydrates, if the body has not enough of one or the other for fuel.

(*b*) *Non-nitrogenous Foods.*—These are derived (*a*) from fats, (*b*) from carbohydrates and (*c*) from vegetable or organic acids. The fats derived from the animal or vegetable kingdom, such as butter, butterine, lard, ham, bacon, olives, olive oil, cream, cheese, pecan nuts, walnuts, hickory nuts, etc., apart from aiding in the reconstruction of fatty tissue, undergo oxidation—*i.e.*, they are burnt up in the body and thus supply heat and energy. The carbohydrates, such as starches and sugars, whatever their source (potatoes, cereals, bread, syrups, fruits, sweets, etc.) enter the blood as sugar. The sugar in the blood is carried to the tissues, where it is burned up, yielding heat and energy. The vegetable or organic acids, like tartaric, malic, citric, acetic, oxalic and lactic acid, existing in fresh vegetables, fruits, meats and milk, are transformed in the system into carbonates, and as such preserve the alkalinity of the blood. In the absence of these acids in the diet, the blood becomes impoverished, and scurvy is likely to develop. The cheapest substitute for fruit-acids is vinegar.

2. *The inorganic food stuffs* are mineral salts and water. The mineral salts constitute about 6 per cent. of the body weight and, as certain quantities are daily eliminated, they must be replaced. The phosphates of lime, potash and magnesia contribute largely to the formation of bone, and are also essential for the growth of the nervous system. Iron is required for the red blood corpuscles and coloring matters. The chlorides are the source of hydrochloric acid in the gastric juice, and keep the globulins of the blood and other fluids of the body in solution. Potassium for the blood cells and solid tissues and sodium for the intercellular fluids are essential for the growth and repair of the tissues. The fact that 60 per cent. of the body is composed of water clearly indicates that a sufficient amount must be introduced to make up the loss sustained by its excretion through the lungs, kidneys, skin and feces. It is simply necessary to recall the physiological functions of water in the absorption and assimilation of food, the elimination of waste products, and its rôle as a heat regulator, to appreciate that a deficiency is certain to be followed by injurious effects. The above classes of food stuffs are essential to life. There is a third class known as food accessories, such as tea, coffee, and condiments; these are important as favoring palatability and digestibility.

Amount of Food Required.—It is apparent that sufficient food must be introduced to make up for the wear and tear of the muscular and other tissues, and also sufficient fats and carbohydrates to supply with the protein at least 3500 calories of energy per day, for the muscular and other work the body has to perform. By the term calories we understand the amount of heat required to raise the temperature of a pound of water 1°F.; or if transformed into mechanical power, such as the muscles use to do their work, a calorie represents force which would be sufficient to lift 1 ton, 1.54 ft. high.

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According to Atwater, 1 lb. of protein or of carbohydrates contains 1860 calories, while 1 lb. of fat yields 4220 calories. For many years it was held that a man weighing 154 lb., and performing moderate muscular work, required, during the 24 hours, 118 grams of protein, 56 grams of fat and 500 grams of carbohydrates; while a man performing hard muscular work should receive 145 grams of protein, 100 grams of fat and 500 grams of carbohydrates.

Professor Chittenden's, and Rubner's, more recent determinations indicate that the protein ration may be cut in two, provided, the fats and carbohydrates are introduced in sufficient quantities to bring the full value up to 2500 or 2600 calories. Other authorities, however, believe that it would be unwise to reduce the protein ration below 100 grams, or 3.5 oz. a day, or the total calories of energy below 3500 in 24 hours. The whole subject of dietary standards is still in its infancy and the best we can do is to make estimates which apply to averages rather than to individual cases. In a general way we may conclude that the needs of the economy are influenced (1) by the height and weight of the individual, amounting to a difference of 40 to 50 calories for each kilogram (2½ lb.) in body weight; (2) by the temperament—nervous and excitable persons require more food than those of a phlegmatic temperament; (3) by muscular activity, which involves not only an increased expenditure of carbon but also increased consumption of protein; (4) age, as during active growth there is also a more active metabolism and children consume more for each kilogram of their weight than adults; on the other hand, with advancing years tissue metamorphosis becomes less active; (5) sex influences the amount of tissue consumption only in so far as there is a difference in weight and muscular activity—an exception should be noted in pregnant and nursing women, who doubtless should receive a more liberal supply of proteids; (6) by temperature and climate. The influence of low temperature results in increased oxidation of carbon, hence an instinctive craving for more fatty food and the carbohydrates (sugar and starches) during the winter months and in cold climates. In the summer months, and in warm climates, there is a repugnance for fat and a craving for refreshing fruits and drinks; hence about 40 grams of fat and between 300 and 400 grams of carbohydrates, with a normal protein ration, will meet the requirements.

Cost of Food.—In our sociological study of families in Washington we found that 476 families, with an income of $500.00 or less, expended 43.68 per cent. of their annual income for food; 159 families, with an income of $500 to $600, 43.59 per cent.; 153 families with an income of $600 to $700, 41.40 per cent., and 153 families, with an income of $700 to $800, 40.21 per cent. for food. The question of food, while of importance to all classes in its relation to health and efficiency, is of special significance, from an economic standpoint, in families with limited means. It has been well said that "half the struggle for life is the struggle for food."

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It has been found over and over again, that persons of limited means purchase food containing little or no nutriment, or select needlessly expensive kinds of food, or prepare a diet altogether too one-sided, and lastly, know little or nothing about the art of cooking. They thus impair not only the nutritive value of the food, but also the digestive functions and general health as well.

In order to give housekeepers of moderate means an opportunity to select suitable dishes I requested Prof. C. F. Langworthy, Expert in Nutrition of the U. S. Department of Agriculture, to prepare for the President's Homes Commission, a chapter on "good food at reasonable cost." He also prepared a table for the use of housekeepers who desire to estimate the nutritive value of the food they prepare. The table shows the protein and energy value of the portions ordinarily served, of the more common articles of food.

This table may be conveniently used by supposing that the food eaten by one member of the family will represent in character and amount the food for the entire family. To estimate by means of the table the food value of the diet, the portions of each article used at each meal should be set down in order, together with the protein and energy which each supplies and the total sum of the protein and of the energy will represent the amount eaten in the whole day.

The question of adequate and proper food for persons of limited means is most important in the present state of high cost of living, and requires judgment and experience, which should be obtained in our public schools. The foundation of a suitable diet will be found in meat milk, butter or butter substitutes, bread, cereals, rice and potatoes. The amount of cooked meat need not exceed 5½ oz. per adult, or about 7 oz. of fish. The daily requirements of fat are about 1 oz. of butter or oleomargarine, or 1½ oz. of bacon—according to the taste and circumstances of the individual. Codfish, herring, milk, butter milk, cottage cheese, peas, beans and lintels are the cheapest sources of protein, and can replace the more expensive meat and egg ration. Bread is a cheap source of both starch and protein and has long been called the staff of life. Oysters, canned vegetables, except beans, and fruit, contain very little nourishment in proportion to their cost. A mixed diet composed of about one-sixth of meat, or meat substitutes, three-sixths of bread and cereals, and two-sixths of fresh vegetables and potatoes, with sugar, syrups, fruit or vinegar, and the fats already referred to, will be found both healthful and economic.

Food should be properly cooked, bills of fare frequently changed, and relishes and flavors should not be wanting. It has been shown that the primary secretion of true gastric juice is the result of a reflex, starting in the mouth, and that the relish of the food originates the reflex. This explains why certain dishes, however inexpensive, "make our mouths water," and why distasteful food fails to cause gastric secretion. Every meal should be a feast. It is a good rule to eat slowly, to masticate thoroughly, and to avoid

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APPROXIMATE WEIGHT AND NUTRITIVE VALUE OF AN AVERAGE PORTION OF SOME COMMON FOODS

Kind of food	Average wt. of portion		Average bulk of portion	Protein	Energy
	Grms.	Ozs.		Grms.	Cal.
1 slice of roast meat	85	3		13	197
1 portion of meat stew	325	11	Saucerful	32	461
1 Frankfurt sausage	60	2		11	170
1 pork chop	190	7		46	765
1 slice of boiled bacon	100	3½		15	432
1 portion of fried bacon	50	2		11	252
1 portion of steak	100	3½		26	411
1 portion of meat soup	190	7	Cupful	7	50
1 portion of pea or bean soup	190	7	Cupful	7	70
1 cup or glass of milk	225	8	½ pint	7	170
1 cup or glass of skim milk or butter milk	225	8	½ pint	7	85
1 portion of cream	65	2	½ gill	2	130
1 egg	50	2		7	96
1 portion of butter	10	½	1-in. cube		95
1 portion of cheese	20	2–3		5	94
1 baked or boiled potato	140	5	Med. size 3 in. long	4	145
1 portion of turnip, beet, carrot, or similar vegetable	130	4½	Saucerful	1	80
1 ear of green corn or 1 portion of stewed corn	75	3	Saucerful	2	82
1 tomato or 1 portion of stewed tomato	95	3½	Saucerful	1	25
1 serving of cooked spinach, cabbage or other green vegetable	125	4½	Saucerful	2	40
1 portion of baked beans or black-eyed peas	200	7	Saucerful	16	300
1 slice of bread	50	2	4 × 4 × 1 in.	4	175
1 portion of corn bread	50	2	3 × 3 × 1 in.	4	142
1 slice of cake	50	2	4 × 2 × 1 in.	3	190
1 slice of apple or other fruit pie	150	5	⅙ of a pie	6	440
1 cup of flour	225	8	½ pint	19	950
1 teaspoonful of sugar	10	½			40
1 cup of sugar	225	8	½ pint		890
1 portion of sirup or molasses	75	2½	About ½ gill		200
1 portion of cooked cereal	200	7	Saucerful	4	170
1 portion of dry ready-to-eat cereal	50	2	Saucerful	6	200
Milk for cereal	25	1	½ gill	1	40
1 portion of boiled rice	140	5	Saucerful	4	155
1 portion of rice pudding, bread pudding, or similar custard pudding	175	6	Saucerful	7	300
1 portion of cherry roll or similar pudding	175	6	Slice	8	460
1 apple or pear	100	3½			50
1 banana	100	3½			70
1 orange	125	4½			50
1 peach or 2 plums	75	2½			35
1 portion of stewed prunes	100	3½	Saucerful	1	115
1 portion of preserves	65	2¼	Saucerful		150
1 portion of fresh berries	100	3½	Saucerful	1	35

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overeating, especially of meat and eggs. Hot and cold food and drinks hastily swallowed should be avoided. (See Teeth, page 466.)

From our knowledge of the proximate constituents contained in various food stuffs, bills of fare can be constructed which will meet the requirements of the body as well as the purse of the consumer.

Miss E. M. Cross of the McKinley Manual Training School prepared for the President's Homes Commission suitable bills of fare, for summer and winter use, for a family of moderate income, which while inexpensive are equal in food value to the best hotel fare.

MENUS FOR WINTER MONTHS

MONDAY

Breakfast.—Hominy, skim milk, cream cake, toast, butter or butterine, coffee. Protein, 28 grams, Energy, 1053 calories.

Dinner.—Irish stew with dumplings, boiled rice, cold slaw, apple pie. Protein, 54 grams, Energy, 1711 calories.

Supper.—Cottage cheese, bread, butter, molasses, tea. Protein, 13 grams. Energy, 818 calories.

Total Protein, 95 grams; total energy, 3583 calories.

TUESDAY

Breakfast.—Rice cakes (left over rice), kidney stew, entire wheat bread, coffee. Protein, 44 grams. Energy, 1176 calories.

Dinner.—Corned beef, boiled potatoes, spinach, tapioca with milk and sugar. Protein, 28 grams. Energy, 842 calories.

Supper.—Fried mush, cold corned beef, bread, butter, tea. Protein, 29 grams. Energy, 1196 calories.

Total Protein, 101 grams; total energy, 3214 calories.

WEDNESDAY

Breakfast.—Stewed prunes, meat cakes, corn bread, butter. Protein, 23 grams. Energy, 771 calories.

Dinner.—Split pea soup, braised beef's heart, boiled cabbage (corn beef liquor), boiled onions, potatoes, apricot roll, vanilla sauce. Protein, 56 grams. Energy, 1572 calories.

Supper.—Corned beef hash, bread, butter, tea. Protein, 29 grams. Energy, 1002 calories.

Total Protein, 108 grams; total energy, 3345 calories.

THURSDAY

Breakfast.—Rolled wheat, skim milk, Potomac herring, corn bread, butter, coffee. Protein, 26 grams. Energy, 866 calories.

Dinner.—Salt pork, potatoes, turnips, escarolle, apple butter, short cake. Protein, 61 grams. Energy, 1530 calories.

Supper.—Pigs' feet, potato cakes, bread, butter, coffee. Protein, 23 grams. Energy, 840 calories.

Total Protein, 110 grams; total energy, 3236 calories.

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MENUS FOR SUMMER MONTHS

FRIDAY

BREAKFAST.—Corn flakes, skim milk, salt-water trout, corn dodgers, coffee. Protein, 28 grams. Energy, 896 calories.
DINNER.—Stewed tripe, boiled potatoes, stewed onions, raw tomatoes, bread, rice pudding. Protein, 37 grams. Energy, 1175 calories.
SUPPER.—Beef stew, corn cakes, butter, stewed apples, tea. Protein, 41 grams. Energy, 1035 calories.
TOTAL PROTEIN, 106 grams; total energy, 3106 calories.

SATURDAY

BREAKFAST.—Fried tomatoes, bacon, bread, butter, coffee. Protein, 31 grams. Energy, 1054 calories.
DINNER.—Boiled leg of mutton, boiled rice, green corn, summer squash, bread, gingerbread. Protein, 32 grams. Energy, 1014 calories.
SUPPER.—Cottage cheese, baked potatoes, raw onions, bread, butter, gingerbread, tea. Protein, 25 grams. Energy, 1048 calories.
TOTAL PROTEIN, 88 grams; total energy, 3116 calories.

SUNDAY

BREAKFAST.—Boiled eggs, Potomac herring, corn bread, butter, coffee. Protein, 35 grams. Energy, 818 calories.
DINNER.—Chartreuse of mutton, tomato sauce, boiled potatoes, string beans, blackberries, milk. Protein, 44 grams. Energy, 1187 calories.
SUPPER.—Rice muffins, baked tomatoes, apple butter, coffee. Protein, 41 grams. Energy, 1066 calories.
TOTAL PROTEIN, 120 grams; total energy, 3101 calories.

THE ALCOHOL HABIT

The habitual use of immoderate doses of alcohol cannot fail to produce serious injury to mind and body. One of the most constant effects is chronic inflammation of the stomach, with consequent impaired digestion and nutrition. Alcohol in excess also produces fatty degeneration of the heart, liver, kidneys and arterial coats, and is one of the most common causes of apoplexy chronic meningitis, insanity and affections of the nervous system, such as inflammation of the nerves, palsies, epilepsy, etc.

It is a lamentable fact that while the mortality from the so-called preventable diseases has markedly declined in the last three decades the death rates from the diseases referred to are very much greater than in 1890. This is justly attributed to the ever increasing per capita consumption of alcoholic beverages.

Every physician knows that the alcohol habit not only predisposes to tuberculosis, pneumonia, typhoid fever and other infectious diseases, but also that these diseases are more fatal or run a more severe course in alcoholic subjects, probably because of the impaired digestive functions and a general

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depraved nutrition of the system, with consequent diminished power of resistance.

Alcohol also increases the susceptibility to industrial poisons, and a tendency to rheumatism and gout. According to Babcock "there is a distinct relationship between the incidence of alcoholism, insanity, venery and crime." The immoderate use of alcohol leads to mental and moral deterioration, characterized by the loss of will power, blunted moral sensibilities, and the ruin of character, and saddest of all, these effects are often transmitted to the offspring.

The experience of 43 American Life Insurance Companies[20] on alcohol in relation to mortality, shows that the mortality of drinkers, who exceed two glasses of beer, or one glass of whiskey daily, was 18 per cent. in excess of the standard of mortality. In those giving a history of alcoholic excesses in the past, the mortality was 50 per cent. in excess of the standard, and in the moderate steady drinkers the mortality was 86 per cent. in excess of the standard.

COMPARATIVE MORTALITY OF MALES 25 TO 65 YEARS OF AGE

All causes complete	All males England and Wales	Brewers	Innkeepers, publicans, spirit, wine or beer dealers
Mortality figures	1000	1361	1521
Diseases of the nervous system	119	144	200
Respiratory system	182	236	217
Urinary system	41	55	83
Liver	39	96	240
Alcoholism	10	25	55
Gout	3	9	13

LIFE INSURANCE TABLES BASED UPON ENGLISH STATISTICS CITED BY JEWETT (TOWN AND CITY, PAGE 87)

Age	Average expectation of life	Moderate drinkers	Total abstainers
At 20 expect to live to be	62	35	64
At 30 expect to live to be	65	43¾	66½
At 40 expect to live to be	68	51½	68

Alcohol as a Cause of Accidents.—Considerable evidence might be adduced to show the relationship of the alcohol habit and accidents. Those who are familiar with the effects of intoxicants need no argument in favor of sobriety. All railroad corporations and industrial plants realize the value of sober habits among their employees, and an increasing number insist upon total abstinence at all times, whether on or off duty.

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Preventive Measures.—The problem of what Hayhurst calls "industrial stimulantism" must be solved by educational methods, and hygiene offers many valuable suggestions. It teaches that intemperance is a vice, the result of a violation of natural laws, and in order to eradicate the evil we must remove the primary causes.

Reference has been made on page 448 to mental and physical fatigue as an important predisposing factor. The indications are to regulate our life so that we do not burn the candle at both ends, take more rest and more interest in general amusements calculated to counteract the influence of saloons. Hygiene has recognized long ago that dust-producing occupations, exposure to extremes of heat and cold, the inhalation of toxic fumes and devitalized air, etc., are fruitful causes of disease in general, and the drink habit in particular, and has also pointed out how the injurious effects may be prevented or at least mitigated. Until this is accomplished by compulsory factory sanitation, pure drinking water, and non-alcoholic beverages —preferably milk—and good nourishing soups should be furnished by the employer.

Every one at all familiar with the subject knows that badly cooked food, especially when consumed from the "cold dinner pail," produces derangement of the stomach and a craving for alcoholic stimulants, which in turn aggravate the original digestive disturbances and readily lead to the drink habit.

In our sociological survey of 1217 families in Washington, D. C., earning less than $1000 a year we found the average expenditure for alcoholic beverages to be $16.14 per annum. Of the 750 wage earners who carried dinner pails 205 consumed alcoholic beverages with their meals.

In addition to the causes mentioned, the unnecessary number of saloons, not infrequently connected with employment agencies or located in the vicinity of workshops, wharves and the homes of wage earners increases the temptation. Last but not least, the characteristic American bar, drinks being consumed in rapid succession, aided by the pernicious system of treating, is a very fruitful cause of the drink habit.

Money spent in temperance saloons for warm wholesome food and soups, cocoa and chocolate, will be a good health investment. It may be stated in general terms that while coffee, tea, spices and condiments in moderation, stimulate the nervous system and increase temporarily the elasticity of mind and body, their abuse is fraught with danger, and we may have "coffee and tea and even coca-cola topers," who suffer from diseases of the nervous system.

It is sincerely hoped that wage earners, in addition to cultivating home life and high ideals, will hold their meetings in special buildings wholly divorced from saloons, and in every way in keeping with the dignity of labor.

The Tobacco Habit.—The per capita consumption of tobacco in the

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United States has increased from 6.31 lb. in 1900 to 7.31 lb. in 1910. In view of the fact that our sociological study of 1217 families in the City of Washington in 1908 shows an average annual expenditure of $12.19 for each family, which at a conservative estimate would amount to $239,655,000 for the annual tobacco bill in the United States, it is important to consider the effects of tobacco on the system.

Tobacco owes its general effects to the presence of toxic alkaloids known as nicotin, nicotellin, nicotein, nicotianin and certain bases such as pyridin, picolin, collidin and others which are formed during smoking. Syrian and Havanna tobacco contain little or no nicotin, while the common grades contain from 3 to 4 per cent. and even 10 per cent. The acute toxic effects of nicotin are familiar to all who have learned to smoke. Strange enough, man becomes accustomed to the toxic effects and may even experience an agreeable excitation of the nervous system, characterized by increased mental and physical elasticity. There is nothing to justify the assumption, however, that the use of tobacco is free from danger. Prof. Seavers' observation on Yale students appears to show that non-smokers made not only the best physical gains in weight, chest measure and lung capacity, but that 95 out of every 100 of the best students were not smokers. His data were apparently so convincing that Japan in 1900 enacted a law prohibiting the smoking of tobacco by persons under the age of twenty. Dr. A. A. Woodhull, U. S. Army, holds that cigarette smoking by the young is harmful as it arrests the natural elimination of waste and hinders the utilization of fresh material, thus interfering with proper growth and nutrition of the body. He also believes that the habit develops a greater tendency to acquire an appetite for alcoholic liquors, premature puberty is induced, increasing the sexual propensity and leading to improper sexual practices. There is a consensus of opinion among educators that the use of tobacco dulls the memory and intellect. When a promising pupil begins to decline in his work it is almost certainly found that he has begun the use of cigarettes. The excessive use of tobacco produces a chronic form of nicotin poisoning, with impairment of vision, nervous irritability or exhaustion, a predisposition to neuralgia and a peculiar affection of the heart, described by Professor DaCosta as the tobacco heart, characterized by irregularity of the heart sounds, accelerated action and weakness of the cardiac muscles. Chewing of tobacco is even more to be deprecated than smoking, as the injurious elements are dissolved by the saliva and not infrequently swallowed.

In many instances tobacco in any form produces a chronic inflammation of the nose, throat and stomach, and acid dyspepsia. Insomnia and blindness, or tobacco amblyopia, are not infrequently observed in heavy smokers. Tobacco smoke also pollutes the air of the room with coal gas and the toxic pyridin bases.

Some authors maintain that nicotin is decomposed during the process of smoking, and that the chief harm results from the pyridin and furfural

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evolved in the smoke. Lehmann,[31] however, recovered in the smoke 82 per cent. of the nicotin contained in cigarettes, and 85 to 97 per cent. in cigars. The London Lancet[32] cites analysis showing from 3.75 to 84 per cent. of the original nicotin contents in the smoke from cigarettes, 77 to 92 per cent. from tobacco smoked in pipes and 31 to 63 per cent. from cigars. The injurious effects of tobacco are also plainly evinced in chewers and in workers exposed to tobacco dust, showing that the nicotin and not the pyridin bases is the chief toxic factor. Kostial[33] has shown that 72 of 100 of the female employees in a Vienna tobacco factory suffered during the first 6 months from congestive headache, palpitation of the heart, precardial anxiety, weakened heart action, intermittent pulse, pain in the stomach, heart burn, vomiting, diarrhea, loss of sleep and appetite, neuroses, general fatigue and loss of strength.

These symptoms combined not infrequently, in acute cases, with faintness, dizziness, pallor of the skin, nausea, vomiting, profuse perspiration and diarrhea, are familiar to all who have learned the use of tobacco in any form, and correspond very closely to those observed in experiments on animals which have also shown that nicotin may produce hardening of the arteries and other degenerative diseases.[34]

Preventive Measures.—The use of tobacco in any form is doubtless a fruitful cause of industrial poisoning among wage earners. This must be apparent when we recall the many ways in which lead, arsenic and other toxic substances may enter the mouth by soiled fingers, chewing tobacco, pipes or cigars which have been contaminated with the dangerous agents. This element of danger has been recognized and in quite a number of European countries the use of tobacco during working hours is strictly forbidden. The employment of chewing gum will not obviate the dangers referred to.

On the whole we may conclude that the use of tobacco is not a physiological necessity, and its abuse, like that of other habit-forming drugs is doubtless a frequent cause of a breakdown. It is especially harmful in nervous subjects and those of insufficient will power properly to restrict its use. A German authority maintains, and no doubt correctly, that the danger is greater from smoking cigarettes, because of the ease with which the smoke is inhaled. In any event, the practice of inhaling the smoke into the lungs, or smoking before breakfast, is a bad one. Pipes, cigar or cigarette holders and the mouth should be kept clean. Symptoms of shortness of breath, obscure pains around the heart, and nervous irritability and cough are indications to reduce or stop the habit altogether.

In a preliminary report on Industry and Tuberculosis, McSweeney and Gunn[35] report that housewives and domestics led the list of occupational classes affected by tuberculosis in their investigation conducted in Massachusetts. Clerks, including salespeople, bookkeepers and stenographers came next. Day laborers, workers on streets, roads, railroads, subway excavations, constituted the third largest class. Miscellaneous factory workers

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in buttons, corsets and typewriters came next. Shoemakers were fifth. Mill operatives, the cotton, hosiery, paper, silk, wool and worsted industries came next. Teamsters, waiters, garment workers, stoneworkers, woodworkers, bakers, metal workers, printers and jewelers also reveal an unduly high tuberculosis incidence (J. A. M. A., June 3, 1916, page 1813).

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CHAPTER I

CHIEF PROCESSES INVOLVING EXPOSURE TO OCCUPATIONAL INTOXICATIONS

SECTION I

PROCESSES INVOLVING EXPOSURE TO ARSENIC POISONING

BY GEORGE M. KOBER, M. D., Washington, D. C.

Mining. Roasting of Arsenical Ore. Manufacture and Use of Arsenious Acid and Color Pigments. Arseniureted Hydrogen Poisoning from Employment of Impure Commercial Acids. Preparation and Use of Ferro-silicon—Preventive Measures.

WORKERS IN ARSENIC

Mining.—Arsenic is usually mined in the form of arsenical pyrites, and when thus obtained, the mining process is believed to be free from danger as regards arsenical poisoning. It has been suggested that the inhalation of dust in mining of an arsenite of cobalt may be responsible for the undue prevalence of cancer of the lungs in the mining district of Schneeberg in Saxony.[1]

Roasting of Arsenical Ores.—Pure arsenic is obtained by roasting native cobalt or arsenical pyrites in special furnaces in the absence of air. Arsenic volatilizes and is condensed on the sides of sheet-iron tubes which are attached to the retorts, and amorphous arsenic oxides and sulphides are condensed in further extension of these tubes which are sometimes made of earthenware. The men engaged in this process and also in the smelting of other ores containing arsenic, such as tin, copper, nickel, lead, iron and silver ore, are liable to suffer from arsenical poisoning, generally in the form of troublesome skin affections. Metallic arsenic is used to impart luster and to harden metal alloys, and in the manufacture of shot to harden the lead.

Arsenious acid or white arsenic is secured by roasting arsenical ores and smelting residues in reverberatory furnaces with free access of air. The arsenical vapors are condensed in suitable chambers and resublimed in iron cylinders. White arsenic is used as a de-colorizing agent in the manufacture of glass, in taxidermy and as an insecticide. The Factory Inspector of East London, cited by Neisser,[2] reported in 1905 a number of cases of skin eruptions in persons employed in packing a powder containing arsenic and to be used in a "dip" for scabby sheep.

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Arsenious acid is also used in the manufacture of colors, such as the green pigment known as Sheeles green, which is an arsenite of copper and Schweinfurth or Paris green which is an acetoarsenite of copper. Other arsenical pigments are used in connection with outside paints, as mordants in dyeing, in the manufacture of colored chalks and for numerous other purposes. Analysis of a sample of dust secured in a room where Paris green was boxed revealed the presence of 0.093 gram of arsenic per cubic meter of air and Dr. Graham-Rogers reports that as a result of spending several days at the plant the mucous membrane of his nose as well as that of Inspector Vogt was inflamed for some time after their visit.[3]

Dr. Alice Hamilton reports cases of ulceration of the skin in the Paris-green factories of Illinois, and states that the workmen are continually shifting

FIG. 30.—Crude method of drying Paris-green. Note dust on walls. (*Illinois Factory Inspection Bulletin*, Vol. 1, No. 1.)

and usually all suffer from arsenical poisoning by the end of the season. In the manufacture of artificial flowers Sheeles green is powdered over the leaves, which constitutes a very objectionable method. According to Morris,[4] from 1 grain to 50 and 60 grains of this pigment have been found per square foot in green wall-paper. This author has also called attention to the fact that stockings, gloves, etc., dyed with aniline colors often cause severe irritation of the skin, because of the presence of arsenic, which in his opinion should never pass into the finished dye, if the process is rightly carried out. Some of the arsenical salts are used in the tanning industry, in etching on brass and bronze, and other industries. See arsenic in the list of industrial

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poisons. It is a remarkable fact that the number of cases of industrial arsenical poisoning, apart from skin affections is comparatively small.

Arseniureted Hydrogen Poisoning.—The majority of cases of industrial poisoning from this gas are caused by the employment of commercial acids, such as sulphuric and hydrochloric acids derived from arsenical pyrites, or the action of such acids upon arseniferous metals.

The inhalation of this gas produces a toxic form of jaundice and hemoglobinuria and other profound symptoms described on page 8.

Prof. John Glaister[5] of the University of Glasgow presented a very important paper on industrial poisoning by gases of arsenic, based upon 120 cases collected by him. These cases have been enumerated by Dr. Legge on page 8.

Of 39 cases of arseniureted hydrogen poisoning reported by Prölss,[6] of which 19 proved fatal within from 3 to 24 days, 12 were chemists, 11 were engaged in filling toy balloons, 7 were aniline workers, 5 lead smelters, 3 were balloonists, and in one the occupation is not stated. Cases have also been reported, apart from the processes already named, in smelter works in the refining of silver from zinc crust with impure hydrochloric acid, and in the formation room of accumulator factories. Three cases occurred in England in an electrolytic process for the recovery of copper.

Rambousek[7] reports five cases which occurred at Breslau in 1902, of whom three died from inhalation of this gas in filling toy balloons. In such instances the hydrogen gas employed is usually generated by the action of sulphuric acid upon granulated zinc or iron which frequently contains arsenic. The cases reported as having occurred in paper-hangers wall scrapers and even in occupants of rooms decorated with arsenical wall-paper are in part caused by arsenical dust, but the cases here reported, clearly refer to the inhalation of hydrogen arsenide gas, evolved by the growth of moulds in starch paste acting chemically on the arsenical salt.

Ferrosilicon.—Prof. Glaister[5] reports a series of interesting cases of poisoning caused by the combined action of arseniureted and phosphureted hydrogen during the transportation by sea or water ways of ferrosilicon. This material is an alloy of iron and silicon and is chiefly used for the manufacture of electrodes and in the production of steel, and thousands of tons are annually shipped from France and Germany to Great Britain and the United States. It is also manufactured in some parts of this country. Ferrosilicon is prepared by melting together iron ore, or iron or steel turnings, with coke, lime, sand or quartz at a temperature of 2000°C. in electrical furnaces. The coke reduces the quartz and ore to silicon and metal with the production of ferrosilicon. Iron and quartz often contain arsenic and phosphorus as impurities, which are not removed, but are converted in the presence of carbon from the coke into calcium phosphide and calcium arsenide. The grades containing 30 per cent. or less, or over 70 per cent. of silicon are comparatively innocous, but those containing between 40 and

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60 per cent. are liable to decompose on exposure to air, especially in the presence of moisture, with the evolution of arseniureted and phosphureted hydrogen. These gases are not only fatal to animals when present in the proportion of 1/4 to 1 per cent., but have also given rise to explosions. Most of the cases of poisoning have so far occurred in confined air spaces on board of ships and canal boats.

The symptoms usually develop quite suddenly, and are characterized by general and abdominal pains, nausea, vomiting, diarrhea, hemorrhages, loss of consciousness, dilated pupils, cold clammy perspiration and death from coma. Cases of poisoning have been mistaken for cholera, gastroenteritis, ptomaine poisoning and pneumonia.

In January, 1905, 50 steerage passengers on the S. S. "Vaderland" en route from Antwerp to New York, and lodged over the hold containing a consignment of ferrosilicon, were seized with an obscure illness and 11 died. The deaths were certified to have been caused by pneumonia, but on account of suspicion of plague, the ship was quarantined until further investigation led to the conclusion that toxic gases evolved from the ferrosilicon caused the illness and deaths. In October, 1905, Dr. Robertson reported similar cases which occurred on a canal boat in England, whereon part of the cargo consisted of ferrosilicon. The brief history given by Glaister shows that the whole family on board suffered from a feeling of sickness and giddiness on getting up in the morning after leaving the port, that these symptoms passed off during the day, but reappeared next morning, and in addition there was also pain in the body, headache and vomiting. On the third day the symptoms were about the same in the parents, but had assumed a very grave aspect in the two children, aged 3 and 4 respectively. Dr. Robertson was called and on arrival found one to be dead and the other unconscious, pupils somewhat dilated, skin covered with a cold clammy perspiration, pulse almost imperceptible, respiration slow and shallow, with crepitant rales all over the chest. This child also died soon after his arrival. Post-mortem examination revealed nothing beyond congestion of the lungs, and analyses of the viscera showed no trace of poison.

In December, 1908, the deaths of five Jewish emigrants during their voyage from Antwerp to Grimsby on the Steamer "Aston," first attributed to cholera and then to ptomaine poisoning, were finally traced to the gases of ferrosilicon, of which the ship carried 9 tons. In another instance cited by Glaister four patients died on a Swedish steamer from the same cause. For treatment see Arsenic and Phosphureted Hydrogen, page 12.

Preventive Measures.—In the prevention of injurious effects, much may be done by the substitution of harmless colors for arsenical pigments. Until this is accomplished special attention should be paid to the employment of wet prcesses; so, for example, the dusting of green arseniferous pigments, in the manufacture of leaves for artificial flowers, etc., from a dredging box is wholly unjustifiable. In occupations where exposure is unavoidable the

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hands should be protected with rubber gloves and the air passages by the use of respirators. Strict cleanliness of the skin and clothing are essential and the rules for prevention of lead poisoning are applicable here. The possibility of arseniureted hydrogen poisoning in the various occupations referred to emphasizes the fact that such operations should never be carried on in confined spaces without the aid of efficient mechanical ventilation. The manufacture of the salts of iron and zinc should be carried on in closed chambers connected with exhaust flues. In the employment of hydrogen gas every effort should be made to secure pure reagents and to avoid close proximity to the communicating nozzle and escape valves, and to make sure that the valves and hose are not leaky.

Rules governing the transport of *ferrosilicon* were formulated in September, 1910, by the Prussian Government. It is required (1) that ferrosilicon be packed in strong water-tight cases of wood or metal; (2) that the words, "ferro-silicon," "to be kept dry," "with care," be legibly and indelibly inscribed on the case; (3) that the substance be delivered dry and in dry cases; (4) that the cases be stored in airy places on the deck of the ship in such a manner that they are protected from wet.

Dr. Copeman, Medical Inspector of the British Local Government Board, suggests the following international regulations for the ocean transport of ferrosilicon. 1. Ferrosilicon should be first broken up into pieces of the size in which it is usually sold, and should be stored under cover, but exposed to the air for at least a month before shipment.

2. Manufacturers should be required to mark in bold letters each barrel or parcel of ferrosilicon with the name and percentage grade (certified by chemical analysis) of the material; the name of the works where it is produced; the date of manufacture; and date of despatch. 3. The shipment of ferrosilicon on vessels carrying passengers should be prohibited. When carried on cargo boats it should, if circumstances permit, be stored on deck. If it be considered necessary to store it elsewhere, the place of storage should be capable of being adequately ventilated, and such place of storage should be cut off by airtight bulkheads from the quarters occupied by the crew of the vessels. 4. This regulation should apply to the transport of ferrosilicon on river or canal barges as well as on sea going vessels. 5. Storage places at docks or at works where ferrosilicon is used should have provision for free access of air, and should be situated at a distance from workrooms mess rooms, offices, etc.

A Committee of which Dr. Copeman was Chairman investigated the accident on the steamer "Aston" and stated, "the proprietors of iron and steel works making use of ferrosilicon will assist in the protection of their work-people and at the same time act for the public benefit by restricting their orders to grades of this material, either not exceeding 30 per cent., or of 70 per cent. and upward, according to the special nature of their requirements."

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SECTION II

PROCESSES INVOLVING EXPOSURE TO BRASS, COPPER (?), ZINC AND OTHER POISONING

BY E. R. HAYHURST, Columbus, Ohio, AND GEORGE M. KOBER, Washington, D. C.

Founding and Casting. Various Poisons. Preventive Measures. Cleaning, Smoothing, Polishing and Buffing Brass Castings. Manufacture of Professional and Scientific Instruments. Manufacture of Electrical Machinery, Apparatus and Supplies. Storage and Dry Batteries. Incandescent Lamps. Manufacture of Bronze Powder. Gilds. Workers in Junk. Mortality Rates.
Copper Mining and Smelting. Work of Coppersmiths. Preventive Measures.
Zinc Mining and Smelting. Galvanizing. Manufacture of Zinc Oxide.

BRASS INDUSTRIES

BY E. R. HAYHURST, Columbus, Ohio

Founding and Casting.—In the brass *foundry* the metal is poured into sand moulds according to certain patterns. In *brass casting* the metal is poured into iron moulds in the shape of bars, tubes or rods which are subsequently rolled or drawn out into sheets, tubes or wire. In both instances the brass is melted, as a rule, in crucibles which are heated each by itself in a submerged cylindrical furnace, although many types of furnaces, less objectionable from a health point of view, are beginning to be used. Scrap metal is dumped into the crucible first, then copper. Just before removing the crucible from the furnace (a period of about 3 hours from the beginning), the zinc ingots are shoved well under the surface of the molten copper mass and, after removal of the crucible, but just before pouring into the moulds or casts, lead is usually added. The workmen are divided into furnacemen, moulders and casters, coremakers, grinders and laborers. Moulders and casters are skilled men except in cases where machines are used. In America unskilled men are largely Eastern and Southern Europeans. Coremakers are often foreign women, who work in separate rooms from the foundry and make fair wages at piecework. During melting and pouring, zinc fumes arise, and, unless provisions are made for confining or removing them, they fill the atmosphere and are inhaled by all those in the vicinity.

These fumes cause the affliction called "brass founders' ague," or "zinc ague" (see Part I). Other immediate afflictions of brass founders are: (1) lead poisoning, usually from auxiliary processes; (2) nausea, vomiting and mucous gastritis from sickening oil furnace fumes; (3) heat prostrations and heat colics due to grave metabolic disturbances; (4) gassing; (5) fatigue and strain; (6) rheumatism and lumbago; (7) acute bronchitis; and (8) industrial alcoholism (see zinc smelterers).

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Various Poisons.—The investigators of the Ohio Survey reported exposure to the following industrial poisons in brass foundries: zinc fumes, lead, antimony, carbon monoxide, carbon dioxide, sulphur dioxide, benzine, phosphorus and "salamander gases."

Preventive Measures.—The brass industry needs a close scrutiny and very material improvement in all branches of industrial hygiene before

FIG. 31.—Casting yellow brass. When the molten metal is poured into the moulds, fumes of zinc arise and are precipitated by the cool air of the room into fine gray powdered flakes. In the room shown these are unavoidably inhaled by the workmen who are thus exposed to the disease known as brass founder's ague. (Photo taken by W. C. Hanson for Mass. State Board of Health.)

workers so engaged can be freed of the risks of degenerative diseases and an abbreviated span of life.

A well-arranged foundry[3] is a roomy, one-story building or top floor, with impervious flooring, ceiling vents, large windows and unhampered by high surrounding eminences. It is divided into: (1) furnace room, or at least partial partitions provide adequate up-and-down ventilation in the furnace

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area; (2) casting room, or an area covered by broad spreading exhaust hoods beneath which pouring is done; (3) moulding shop, where moulds are made (an entirely cold process); (4) core room, which has the core-baking ovens well apart; (5) grinding and sand-blasting rooms, equipped with exhaust ventilation, where rough castings are smoothed down; (6) change house, or quarters provided with lockers on one side for street clothes, shower baths

FIG. 32.—Casting yellow brass. Brass workers are here protected from zinc fumes by an adequate exhaust system. (Photo taken by W. C. Hanson for the Mass. State Board of Health.)

in the center, and lockers for work clothes on the other side; (7) luncheon quarters. At present, a very few foundries in America approach this standard. In most instances all or nearly all the above processes are carried on in the same room, often low-ceilinged, while doors and windows with perhaps a few small openings in the roof—all of which are closed in the winter time—are depended upon for ventilation.

Furthermore, prophylaxis consists in selecting workmen, regulating their

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habits and work, then limiting, confining or removing the fumes. The adoption of the electric furnace promises much in this respect. Aerial dilution of the fumes is not enough. All furnace areas and pouring areas should be provided with hoods and stacks to draw off vapors. The Germans are using flexible exhaust ducts which are locally applied during pourings.[4] In addition, the foundry rooms need air-agitators (fans), and arrangements for vertical (floor-ceiling) ventilation which is most apt to be efficient irrespective of weather and wind conditions. The wearing of respirators although a help will not prevent the inhaling of gas or fumes. The more zinc oxide in the air the more imperatively are regulations and improvements needed. Foreign countries have adopted extended regulations for smelting and founding industries and marked results have already been produced according to their statistics.[5]

Courtesy of Crane Co.

FIG. 33.—Crucible of yellow brass. Approved method of handling. (*Illinois Factory Inspection Bulletin*, Vol. I, No. 1.)

Fortunately the vast majority of brass workers are employed outside of the foundry in finishing processes, such as polishing and buffing (very dusty work, but in the past 10 years rendered practically harmless by exhaust systems locally applied), machinery and assembling work, plating, lacquering, etc. Among assemblers of brass parts, as chandelier makers, lead poisoning occasionally occurs due to the careless use of white or red lead paste in hermetically sealing joints.

Cleaning and Smoothing of Brass Castings.*—This process is accomplished in several ways. One method is by chipping the rough castings

* By George M. Kober.

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with pneumatic chisels or by means of "sand blasting." In some of the factories the smaller castings are cleaned by hand with steel wire brushes, or with mechanical revolving wire brushes, or by being mixed with small pieces of scrap iron and then rotated in "tumblers" or "rattlers." The constant friction of the castings upon each other and the scrap iron removes the sand and all rough surfaces.

Polishing and Buffing.—This is usually accomplished by the employment of emery wheels and the final burnishing of brass is done on "buffing wheels"

Fig. 34.—Buffing brass. The buffing wheels shown are well equipped with hoods and blowers and give off practically no dust or lint. (Supplied by W. C. Hanson for the Mass. State Board of Health.)

covered with leather or circular pieces of cotton cloth. All of the processes referred to are extremely dusty and expose the operatives to the inhalation of a conglomeration of dust and also to injuries of the eyes.

A number of cases of plumbism are annually reported among the polishers or buffers of brass goods, especially of spiggots. Gun and pot metal contain usually from 5 to 6 per cent. of lead, but the Medical Inspector of Great Britain[6] has reported instances of lead poisoning in brass polishers in which analyses revealed the presence of only 2.4 to 2.9 per cent. of lead. The factory inspector of the State Board of Health of Massachusetts[7] also points out that many of the brass polishers are pale and emaciated, their teeth in

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a bad condition and the gums showing the characteristic bluish discoloration. Dr. Wm. Murray[8] was the first to point out that the cases of lead poisoning in "chandelier makers" are contracted by the habit of fitting the joints with lead and then testing them by closing one end and sucking the other end of the bracket.

As in the case of copper goods a dip of dilute sulphuric acid is quite generally used and naturally adds to the danger. The use of japan mixed

FIG. 35.—Buffing brass. No dust-removal device is here provided for the protection of employees. (Supplied by W. C. Hanson for the Mass. State Board of Health.)

with *wood alcohol* and of *Zapone lacquer* (amyl acetate) as a final finish to chandeliers, art goods, etc., is injurious to the health and calls for special protection in the way of exhaust ventilation and steam-heated drying chambers. (See page 721.)

Instruments, Professional and Scientific.—The most important unhygienic processes involved in the manufacture of such instruments are those already referred to, such as machine shopping, soldering, welding, polishing, buffing, electroplating, etc. In the manufacture of optical lenses, microscopes, etc., there is also exposure to dust from glass and rouge (iron peroxide) during the grinding and polishing processes, and in the manufacture of barometers and thermometers there is exposure to lead and mercury.

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Makers of *muscial instruments* often acquire lead poisoning by filling the instruments with molten lead before the shaping process.

Electrical Machinery, Apparatus and Supplies.—The manufacture of electrical switch boards, meters, telephones, registering devices, etc., involves hazards common to the brass industry, except that the workers on electric meters and electrodes are also exposed to mercury.

Storage Batteries.—The occupational risks in the manufacture of storage batteries, from lead poisoning, have been referred to on page 115. The dangerous process consists in the casting, dressing and polishing of perforated or grooved lead plates, the connection of these plates by the use of solder and blowpipe and the employment of lead oxide paste and sulphuric acid. The rapid solder employed in storage battery work also contains mercury. In many establishments fully 50 per cent. of the workers have been known to contract lead poisoning, some of them within 10 days after beginning the work.

Dry Batteries.—The manufacture of dry batteries involves exposure to a number of industrial poisons, such as benzol, creosote, hydrochloric acid, lead during the soldering process, mercury during the amalgamizing process, pitch and zinc chloride. Inflammatory conditions and fissures of the skin and chloride of zinc and acid burns are not uncommon. Cancer of the skin, attributed by some authors to carbon dust, the use of pitch and chemicals, has been reported.

Incandescent Lamps.—One of the chief characteristics of this industry is the overwhelming number of female employees and the "speeding-up" incident to piecework. Much of the work is done in close quarters and involves exposure to industrial poisons. Commendable progress has been made, however, and the Ohio Survey reports that none of the employees were exposed to mercury, which was used in closed containers to produce a vacuum, and that red phosphorus was used instead of the white or yellow variety in painting. The chief hazards in each of the different departments found by Hayhurst were as follows: *Filament making:* wood alcohol, gas fumes, heat, inadequate ventilation. *Tubulating, sealing* and *painting:* the hazards already mentioned, also exposure to glass dust, foreign bodies in the eyes, small cuts and burns. *Vacuum production, photometry* and *aging:* gas fumes, heat, eye strain from bright and flashing lights. *Finishing processes:* gas and solder fumes, wood alcohol, some eye strain, troublesome calluses of hands, caused by the use of wire cutters. Amyl acetate is used in some establishments as a lacquer.

Bronze Powder.—The manufacture of gold, silver, bronze and copper powder is attended with the evolution of considerable quantities of very fine dust even though the process of grinding, powdering and drying is carried on in inclosed machinery.

The ordinary gold bronze powder is composed of copper and zinc, with traces of lead, tin, arsenic and iron. Simon and Knyvett[9] call attention

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to the possibility of lead poisoning because in a case brought to their notice the bronzing powder contained 7 per cent. of lead. Popoff, cited by Casamajor,[10] described a case of general muscular atrophy and gastro-enteric symptoms in a bronze worker which he attributed to zinc poisoning. Quite a number of persons engaged in handling bronzing powder complain of a coppery taste in the mouth, but not many cases of plumbism have been reported. Catarrhal affections of the eyes and respiratory passages, headache, and gastric derangements are not infrequent and Oliver[11] refers to the process of "dry bronzing" as a cause of inflammatory conditions of the skin. On the other hand, Wollner[12] maintains that the bronze workers at Fürth are a comparatively healthy class. Bronzes are often suspended in benzene, acetone, pyroxylin, wood alcohol, amyl acetate, etc., with consequent menace to healths. (See also pages 614 and 677.)

Gilds.—Analyses by the Ohio State Hygienic Laboratories[34] of six leaf gilds (gold, silver, copper, aluminum and white finish) made by several manufacturers failed to show the presence of either lead or arsenic.

Workers in Junk.—In a number of cities special establishments exist where the process of sorting rags, paper and metal is carried on in large sheds and warehouses, and where the non-ferrous metals are melted down. The various processes are usually carried on under the most insanitary conditions and are doubtless inimical to health. Hayhurst in his Ohio Survey reports four cases of lead poisoning and also believes there is considerable risk from brass or zinc poisoning where some of the soft metal alloys are handled and melted. He also reports that several cases of lock jaw had come from one of the large plants.

Mortality Rates.—The death rate from tuberculosis among brass workers is twice that of iron and steel workers.[1] Of 201 brass workers, 31.3 per cent. died of pulmonary tuberculosis, 9.4 per cent. of Bright's disease, 9.0 per cent. of pneumonia, 8.4 per cent. of cancer, 8.0 per cent. of accidents. Hoffman's figures[2] for 1897 to 1906 cover 414 deaths: consumption 38.9 per cent., Bright's disease 9.9 per cent., pneumonia 8.7 per cent., heart disease 6.5 per cent., accidents 4.6 per cent. Hoffman says, "The proportional mortality from this disease (consumption) was excessive at all ages under 65, but most so at 15 to 24, when out of every 100 deaths from all causes 59.1 per cent. were from consumption, against a normal expected proportion of 27.8 per cent." Insurance risks on brass founders, molders and casters are about 12 per cent. higher than normal. Those in finishing processes, including grinders and polishers, have more privileges or are not discriminated against at all.

COPPER INDUSTRIES

Mining.*—*Copper miners* and those who handle ores, as at milling, are liable to arsenic poisoning and also plumbism, providing lead is present in a

* Prepared by E. R. Hayhurst, Columbus, Ohio.

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soluble form. The ore dust itself produces digestive, respiratory, skin and conjunctival disease. *Miner's boils,*[13] occurring in Michigan mines, are probably due to the action of the hard alkaline character of the water upon the skin, thus predisposing to infection. *Miner's cramps* occur especially among trammers in deep mines, and appear to be due to heat-fatigue toxins. These cramps may involve the whole body, last for hours or days, and perhaps end fatally. The exhaustive character of work in connection with one-man drills[14] is a recent factor of complaint on the part of copper miners.

FIG. 36.—Copper mining. Compressed-air gang at work on face of ore, Butte, Montana. (Photo furnished Würdemann by Dr. Donovan.)

Hook-worm disease is a great factor in older mines, or where foreigners are employed (including Cornishmen), or where temperatures reach over 70°F. In California mines 50 to 80 per cent. are so affected with a loss in efficiency conservatively estimated at 20 per cent. of the working force.[15] According to Hoffman,[16] *mortality rates* among copper miners for 1911 ranged from 3.32 to 3.64 per thousand, with tuberculosis claiming about one-third of the deaths, and accidents a close second. Metal miners and smelterers are declined or considered hazards in life insurance.[17] (For discussion of Copper Poisoning see Part I.)

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Smelting.*—The smelting of the ore is carried on in reverberatory furnaces and involves exposure to sulphurous and possibly arsenical fumes, and also to the products of combustion which sometimes contain 25 per cent. of carbon monoxide. In the electrolytic process of copper refining there is exposure to acid fumes. Hard work, exposure to excessive heat and sudden changes are likewise injurious factors and help to account for the pale and ill-nourished condition of the workers.

In most of the modern smelters steps are taken to utilize the monoxide of carbon for fuel, and the sulphurous fumes are condensed in leaden chambers for the purpose of making sulphuric acid.

(For a full discussion of copper fumes see page 16.)

Preventive Measures.—The dangers incident to the smelting operation can be materially diminished in this as in all other similar processes by high stacks and well-constructed furnaces, proper ventilation and sprinkling of the flues and chambers before they are cleaned out and the employment of respirators by the men engaged in this work. It is equally important that during the pouring of the metal the employees should be protected from the toxic fumes by hooded exhaust ventilation and suitable respirators.

Coppersmiths, etc.—Copper workers show a high mortality from diseases of the respiratory organs and the consumption rate in Great Britain, according ot Dr. Tatham,[18] is 59 per cent. in excess of the average. The mortality from diseases of the circulatory, digestive and urinary system is also greatly in excess of the average among other metal workers.

The men employed in the manufacture of copper cornices, tubing, utensils, ornaments, etc., especially those engaged in filing, grinding, turning and polishing, are more or less exposed to the inhalation of copper dust and metallic oxides, and likewise to the dust from emery and polishing wheels. Quite a number of the workers show a green discoloration of the gums, teeth and hair, and the characteristic green stain of the underwear from the perspiration. Some dentists have reported a peculiar purple color and swelling of the gums with more or less stomatitis, in copper workers. Arlidge and Blaudet[18] have held for a long time that the copper workers are liable to a specific intoxication, characterized by colic, vomiting and purging. Dr. Simon[18] admits the possibility of such effects in men, who by reason of unclean habits are liable to swallow copper dust with their food, especially those who work in old copper and brass covered with more or less copper carbonates.

Zadek[19] on the other hand cites a number of authorities to support the opinion that copper itself is not a toxic agent, and that the symptoms, such as colic, etc., are caused by alloys, chiefly lead, and that the green discoloration of the teeth and hair has no pathological significance. Leurin,[20] while questioning the possibility of chronic copper poisoning, concludes that

* Prepared by George M. Kober.

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acute intoxications may occur after ingestion of considerable quantities of the metal.

Animal experimentation, conducted by Baum and Seeliger[21] would appear to establish, however, the possibility of chronic copper poisoning. Acute bronchial catarrh, rheumatism, gastro-enteric catarrh, tonsillitis, neuralgia, eczema, diseases of the veins, contusions, fractures, burns and wounds and lead poisoning are prevalent affections among the coppersmiths of Berlin. The average age of the time of death was 47.4 years.[22] Coppersmiths, like boiler makers, are liable to develop progressive deafness. Cases of oxalic acid poisoning contracted during the polishing of copper or brass utensils have been reported. The men engaged in soldering of copper are exposed to lead and the inhalation of acid and possibly arseniureted hydrogen fumes. Copper is also used, in connection with gold, silver and mercury, in water gilding and in the manufacture of bronzing powder. The sulphate of copper is used for coppering wire. Many of these processes involve not only the inhalation of dust, but also of acid fumes and therefore engender diseases of the respiratory and digestive organs.

Preventive Measures.—Much of the danger of dust inhalation has been eliminated by the substitution of acid dips for polishing but the acid dipping solutions expose the operatives to the inhalation of irritating fumes and hence all operations involving the evolution of dust or fumes should be carried on with adequate provisions for hood and exhaust ventilation.

ZINC INDUSTRIES*

Mining.—*Zinc miners* are liable to arsenic and "metallic" poisoning,[23] and, where the ores contain manganese, those exposed to the dust have developed peculiar nervous symptoms.[24] American miners are very little liable to lead poisoning in connection with zinc mining and ore milling, since the lead is in the insoluble form, as a rule.[25]

Smelting.—American ores are usually smelted by the Belgian process in which the ore is first roasted to drive off the sulphur (sulphuric acid is an important by-product), then volatilized in clay retorts having muffles attached in which the fumes condense. The escape of zinc fumes which sometimes contains also the fumes of antimony, arsenic, cadmium, lead and manganese is rendered almost negligible. Zinc smelting has certain forms of occupational complaints: (1) "zinc chills" (see Part I) which are of infrequent occurrence because from industrial economy the escape of fumes is limited; (2) lead poisoning, where lead occurs in the ores, is of greater frequency than among brass moulders (the lead burners or solderers who work in the acid tank houses are also very liable to plumbism); (3) "summer colics," probably due to heat-fatigue toxins, plus the drinking of cold water; (4) heat prostrations; (5) "zinc asthma," which is very prevalent and shows winter exacerba-

* Prepared by E. R. Hayhurst, Columbus, Ohio.

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tions; (6) gas poisoning, from the producer-gas used; (7) industrial alcoholism, due to a tradition among the workers that alcoholic liquors are antidotal to zinc and other metallic poisonings, but heat, dust, gases and fatigue also contribute to it; (8) rheumatic affections—very common—especially lumbago; (9) dermatitis and boils, due to the ore dusts; (10) a rare possibility of arsenic poisoning, in the American works; and (11) night blindness,[26] probably a zinc effect, but may be due to looking at zinc flames. It contributes to accidents. Colored glasses should be worn by furnacemen.

Rambousek's[27] summary of the afflictions of zinc smelter workers shows that among 4789 men there were 50 cases of lead colic, 18 of kidney diseases, 223 of gastric and intestinal catarrh, 40 of anæmia, and 612 of rheumatism in 1 year's time. (For insurance risks see page 499, under copper.) According to Dr. Krautz[28] the number of cases of lead poisoning in the German zinc-smelting industry has been reduced by hygienic methods from 819 cases among 1200 employees in 1879–1885 to 78 cases per 6400 workers in 1910.

Zinc is extensively employed in sheet metal for many purposes, and it is an important constituent of brass. It is also used in the galvanizing process of sheet iron, wire, etc., and in the manufacture of zinc oxide and other zinc salts. According to Tatham the mortality of zinc workers in Great Britain was 1198, as against 602 for agriculturists. Consumption and diseases of the respiratory and circulatory organs predominate.

Galvanizing.—Sheet iron or metal to be galvanized is first cleansed in a "pickling" bath of hot dilute mineral acid, and then dipped into a bath of molten zinc, which is kept from overheating by sprinkling sal ammoniac upon its surface. A layer of melted lead forms the bottom of the zinc bath, its heavier specific gravity keeping it down, where it helps to prevent overheating. The occupational afflictions of this industry may be summed up as follows: (1) Inhalation of acidic steam and vapor from pickling, causing rhinitis, frontal headaches, conjunctivitis, bronchitis, and asthma, dental caries, reddening and softening of the gums, hyperacidity, mucous gastritis, and attendant disturbances, also the immersion of the hands in acid baths whitens and hardens the skin which tends to crack; (2) sal ammoniac "smoke," when inhaled, at first causes coryza, with perhaps epistaxis, nasal sores and dyspnœa, but workmen become rapidly inured to it; (3) rheumatism and lumbago, from alternations of heat and cold exposures, especially when combined with occasional heavy lifting or straining; (4) dermatitis, from the chemicals used; (5) arsenic poisoning as a rare possibility, from the spelter; (6) "zinc chills"[29] (see Part I), which can occur only when the metal becomes overheated; and (7) lead poisoning, infrequently and probably due to handling the metal rather than from any escape of lead as fumes.

Manufacture of Zinc Oxide.—Ores of second grade, as a rule, are smelted and the zinc fumes condensed in long air-cooled pipes under negative pressure, which convey the zinc oxide formed to bag houses. Here the oxidized coal gases and other gases escape through the meshes of the muslin

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bags, while the flaky oxide is caught. The afflictions of the furnacemen are practically the same as those of smelterers, but zinc chills (see Part I) are less common because of less exposure. However, they may occur.[30] Bassett[31] states that they have been a very serious factor in securing and retaining labor, particularly in the bag rooms. This is entirely a question of the completeness of the oxidation and the cooling of the oxide before it reaches the bags. In the new and model plant which I recently visited at Palmerton, Pa., bronchial afflictions were said to be the only complaint among the bagmen, this from the escaped dust. Packers were more troubled even than bag-house men.[32] Zinc dust (pulverized zinc) and zinc oxide may also produce dermatitis, conjunctival and digestive disturbances, similar to other metallic dusts, and workmen should be protected as well as possible. Zinc dust may be a constituent of "bronze" powders. Zinc oxide may be used in brewing to assist in disinfection and preservation. Zinc white (zinc oxide) is used in great quantities to mix with dried rubber to secure the physical properties necessary for pneumatic tire casings. Zinc white and *lithopone* (the latter is zinc sulphide and barium sulphate) are the best substitutes for lead in paint. Lithopone makes an excellent marine paint. Sir Thomas Oliver[33] says that zinc can certainly be used for all interior decorations, and that reformers maintain that the slight advantage of white lead over zinc for exterior painting is not worth the cost to human life.

Robinson and Wilson[35] inspected eight *brass and copper* establishments in Cincinnati and examined 2072 workers inclusive of 142 females and found 17 cases of tuberculosis or 0.88 per cent. among the male workers and none among the females. This is a very much lower rate than in certain other industries. Sand blasting, grinding, buffing and polishing were extremely dusty processes before the installation of the exhaust system, and this hazard has not yet entirely been eliminated.

The investigators comment favorably upon the sanitary conditions in the larger plants, but this cannot be said of all the establishments, as they received a letter from an employee in a brass factory to which admission was refused, requesting an investigation, and stating that the men are often overcome by the fumes in the melting department.

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SECTION III

PROCESSES INVOLVING EXPOSURE TO CARBON BISULPHIDE AND THE INTOXICATIONS IN THE INDIA-RUBBER INDUSTRY

BY E. R. HAYHURST, M. D. AND GEORGE M. KOBER, M. D.

Rubber Washing. Compounding. Mixing Mills. Calendering and Warming Mills. Tire Building. Buffing. Dipping. Manufacture of Rubber Mechanical Goods, Drug Sundries, Accessories, etc. Steam and Vapor Vulcanizing. Cold Vulcanizing. Rubber Cement Manufacture Rubber Reclaiming. Manufacture of Rubber Substitutes. Preventive measures.

THE INDIA RUBBER INDUSTRY

This industry includes all establishments in which waterproof garments, boots, shoes, rubber gloves, water bags, ice-caps, surgical appliances, children's toys, rubber tires, door mats and any other rubber goods are made.

India rubber is the inspissated juice of various rubber plants, and is known as caoutchouc. This is insoluble in water, but quite soluble in ether, acetone, petroleum benzine, benzene, bisulphide of carbon and turpentine.

Benzine is most frequently used as a solvent and serves to convert the rubber into a doughy mass, with which other materials such as white lead, zinc oxide, chalk, litharge, cinnabar, graphite, powdered soapstone, etc., are mixed.

Cases of lead poisoning may occur from the use of litharge or other lead compounds. Of 162 cases of lead poisoning treated in the Massachusetts General Hospital Dispensary, 18, or 11 per cent., were among rubber workers.[1] Of 544 cases of lead poisoning reported by Hayhurst[2] in the Ohio Survey, 43 occurred in the rubber industry.

Cinnabar and antimony are among the red coloring agents used in the manufacture of rubber goods. Roth[3] refers to the fact that French authors some years ago called attention to a peculiar "caoutchouc disease" which they attributed to the cinnabar contained in the gutta-percha plates of artificial teeth. Oliver[4] cites the observation of Lazarus, a German dentist, who reported 20 cases of dental caries in gutta-percha workers, complicated in some instances with necrosis of the jaw bone, but offered no explanation as to the probable cause.

The rubber industry is a very important one in the State of Ohio, and Hayhurst found the chief poisons employed, in the order of their danger to users, as follows: aniline oil, carbon bisulphide, benzol, lead and antimony compounds, mineral acids, alkalis, benzine (naphtha, petrol, gasoline) wood alcohol, sulphur chloride, carbon tetrachloride, mercuric sulphide and turpen-

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tine. In some of the processes there was an additional risk from exposure to illuminating and fuel gas. The following is a summary of Hayhurst's exhaustive report of the different processes involving special hazards.

1. **Rubber Washing.**—This process deals with the cleaning and preparation of the crude rubber, such as cracking it up in mills, washing it in cold and warm running water and subsequent steeping in large tanks of water. This process involves wet and sloppy work and exposure to a steamy atmosphere. In some of the plants lead and antimony were used in dust form in the same room, although quite foreign to this particular process, and with considerable liability to poisoning all the workers.

2. **Rubber Compounding.**—In this department the various dry ingredients which go into the rubber, such as metallic oxides and salts (Al, Fe, Ca, Sn, Zn, Pb, Sb), soapstone, etc., are scooped from bins according to various formulas, weighed by hand, placed in open top tin boxes, and carried direct to the mixing mills or delivered close thereto through pipes, there to be incorporated with the rubber. Occasionally the ingredients are first bolted in shakers to break up lumps, which is an exceedingly dusty process unless the shaker is well inclosed. The work also involves exposure to lead and antimony dust, and to the fumes of aniline oil, which is often poured directly upon the weighed powders from an ordinary cup. In the Ohio Survey 22 cases of positive and 5 cases of tentative lead poisoning were observed among the 151 wage earners employed in this process. Two cases of acute aniline poisoning were reported, but had not been seen by the investigator. Hayhurst believes that the workmen in this department should be subject to careful supervision, monthly medical inspections, and feels confident that the work could be rendered practically dustless, by competent ventilating engineers.

3. **Rubber Mixing Mills.**—In this process the washed and dried rubber is macerated between steel rollers, and mixed with the compounds referred to in paragraph 2. In spite of hoods and exhaust flues, even when present, more or less dust escapes while the injurious ingredients are scooped out of the conveyance boxes and poured by hand upon the rolls. The amount of lead dust is estimated by the Ohio Survey as high as 25 per cent., and the odors from aniline oil and of antimony fumes were plainly detectable in several establishments. The investigators discovered 22 positive cases and 4 tentative cases of lead poisoning, and 3 positive cases of aniline poisoning, among 252 male wage earners. The risks of poisoning were considered bad in 13 out of 21 establishments because of lack of adequate ventilation for the removal of dust and fumes, lack of medical supervision and instructions, and hence ignorance on the part of the workers, many of whom were wearing mustaches and eating and chewing while at work; there was also lack of personal care and of adequate washing facilities.

4. **Rubber Calendering and Warming Mills.**—In this process the compounded material brought from the mixing mills is placed into the rollers of

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the warming mills and of the calenders, which perfect and grade down the material into sheets of required thickness. The calenders are also used to spread and press the rubber mass over the fabric to be waterproofed, which process is called "frictioning." When this process is carried on in the same room with the mixing mills the danger from poisonous dust inhalation is quite great. But even when completely separated there is more or less soapstone dust, which is freely used in the handling and packing of the finished goods. The heat from the rollers is apt to be excessive, unless guarded against by adequate exhaust ventilation. Other occupational risks are exposure to benzine vapors especially from open cement cans. In the Ohio Survey no cases of occupational poisoning were observed in this process, although in only three places were all the workers healthy appearing.

5. **Rubber-tire Building.**—In the manufacture of casings for automobile and bicycle tires different layers of rubber fabric are applied upon wheel-shaped moulds by workers who are seated before them, and when thus built up by hand or machine labor, the beads, cover layers, etc., are finally attached by means of rubber cement and pressure. In this process benzine is freely used with the sponge and also as a solvent for the cement. In 9 of the 13 establishments investigated in Ohio, the odor from benzine was strong enough to cause symptoms. Nine cases of benzine poisoning in female workers in a rubber-tire factory at Upsala were reported, of whom four died. The history does not make clear that they were cases of petroleum benzine or of benzene poisoning.[5]

The chief complaints in the Ohio Survey were headache, dizziness and stupefaction. Antimony dermatitis was an occasional complaint. Soapstone dust was present in variable amounts in all places. The effects of fatigue as a result of speeding up, faulty positions, jarring processes, pressure against the body and lifting and carrying of heavy moulds were evident in most places. In winter the effect of cold drafts on sedentary workers from windows kept open to drive out the fumes was an objectionable feature.

The inner tubes for rubber tires are made by lapping long strips of almost pure rubber around an iron rod of suitable size, or by butting the edges together on a table top. The rubber is made to adhere to itself by moistening with benzine or rubber cement. The long tube is next wrapped with a wet strip of cloth and then steam cured, after which the ends are spliced together, as a rule, by a cold-cure process. This work is performed to a great extent by youthful men and women and involves considerable standing and walking to and fro, but the chief hazards are exposure to benzine and soapstone dust. Benzine stupor and fainting spells were common, especially among females and recent employees.

Copious ventilation, a room temperature not exceeding 68°, and the substitution of closed containers with a pressure-valve benzine emitter, in place of the open cups now used, would greatly diminish the dangers from benzine fumes.

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6. **Rubber Buffing.**—The process of buffing rubber-tire casings is done by revolving them on a wheel and holding a coarse file against them. Other semi-hard rubber objects are buffed by means of emery wheels. The object in either case is to secure a rough surface for the cement, in order to apply additional coats. There is also exposure to dust during the grinding and polishing of hard-rubber goods.

This work, apart from being fatiguing and very dusty and dirty (workers look like coal heavers), involves the risk of lead poisoning, which varies in degree with the amount of lead contained in the rubber dust. Benzine fumes were also present in most of the establishments covered by the Ohio Survey.

7. **Rubber Dipping.**—In this process moulds of gloves, finger cots, etc., are dipped mechanically into vats containing a solution of rubber dissolved in benzine. Upon raising the objects from the vats, a thin coating of rubber adheres to the mould, and the dipping is repeated until the required thickness is secured. The process involves exposure to benzine fumes and great precautions are necessary to avoid fire and explosions. Benzine fumes, from the vats and dipping frames above, constituted a bad hazard in seven places and to a less degree in four establishments covered by the Ohio Survey. The benzine tanks should be covered when not in use, the floor should be latticed, with a strong exhaust system beneath to draw off the benzine fumes, and the entire system of exhaust ventilation should be set in motion during the dipping process.

8. **Rubber Mechanical Goods, Drug Sundries, Accessories, Etc.**—This group includes the manufacture (assembling and finishing) of a great variety of soft- and hard-rubber goods, such as rubber bands, washers, children's toys and balloons, water bags, ice-caps, surgical appliances, drug sundries, finishing of rubber gloves, assembling of boots, waterproof garments, door mats, fire hose, large belts for power transmission, and other rubber fabrics. The Ohio Survey covered 22 establishments with 2912 male and 1668 female employees. The chief health risks were benzine fumes and soapstone dust; occasionally also benzol, sulphur chloride, carbon bisulphide, ammonia, aniline dyes and wood alcohol. Benzine vapors are constantly evolved, but are especially pronounced when rubber solutions are used for waterproofing, in the joining of the seams, in cementing edges, laps and splices, and during the evaporation of naphtha from the finished product, which is done over steam tables. In but one plant were all employees satisfactorily protected from these forms of industrial poisoning. As a matter of fact, 10 cases of chronic benzine (and perhaps benzol) poisoning were seen in four plants, and quite a number of the employees, especially females, suffered from nausea, dizziness, faintness, headaches, loss of appetite, loss of weight, tiredness, "benzine jags," eczema, coughs and colds. Close confinement and crowding of workers are often observed. Sometimes windows are not permitted to be opened for fear of air effects,

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but there is no consistency about this and the recommendations for the removal of dust and fumes already referred to are equally indicated in this branch of the rubber industry.

9. **Rubber Vulcanizing.**—All rubber goods, in order to withstand the influence of changes in temperature, and to retain their elasticity, are vulcanized. This process is also spoken of in the trade as "curing."

Steam Vulcanizing of Tire Casings.—In the comparatively harmless or steam process, the tire casings are placed in iron moulds and these are placed within steam cylinders and exposed to a temperature of 260°F. In some plants the tires are steam-cured when partly built and again when finished. There are no special poison risks, except the escape of fuel gas fumes in some plants, but soapstone dust and the exposure to high temperature and humidity are common observations.

Press Rooms.—In these rooms steam vulcanizing is done by means of steel presses, having hollow chambers to accommodate the various shaped moulds through which the steam passes. The rubber goods, such as water bags, drug sundries, etc., are placed within the vulcanizers for from 15 to 30 minutes. Limited amounts of sulphur compounds, such as chloride of sulphur, sulphide of barium, calcium or antimony, are added to the rubber goods.

The special hazards observed in the Ohio Survey were benzine, benzol and occasionally antimony fumes from the rubber. Exposure to excessive heat (120°F.) humidity, abrupt changes in temperature, and considerable quantities of soapstone dust are mentioned as injurious factors. The generous use of asbestos coverings to the presses and more copious room ventilation are recommended.

Vapor Vulcanizing.—In this process the rubber goods, such as rubber gloves, finger cots, etc., are exposed to the vapors of sulphur chloride, placed upon a tin in a warming cupboard. In some establishments, carbon tetrachloride and perhaps carbon bisulphide are used. In addition to exposure to these industrial poisons, there may also be exposure to benzine, benzol and soapstone dust. The fumes should be withdrawn before opening or entering the cupboards.

Cold Vulcanizing.—This process, also known in the trade as "cold cure" and "acid cure," is chiefly used for joining the ends of inner tubes for tires and also for curing pure rubber goods and is quite dangerous, especially when carbon bisulphide is employed. In this process the rubber goods are passed through a solution containing usually 1:1000 parts of carbon bisulphide and from 2:10–1000 of sulphur chloride and subsequently dried in cupboards at a temperature of about 104°F. Sometimes the solution is applied with a brush or sponge. During this process the employees are exposed not only to the fumes of carbon bisulphide but in the dipping of the goods also to absorption of the poison by the skin. This poison inflicts special damage to the blood cells and general nervous system, and like benzine also causes a dry and

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harsh condition of the skin of the hands by removal of the natural fat. (See carbon bisulphide, pages 35, 725.)

Laudenheimer[6] has collected three cases of mental disturbances and 19 cases of diseases of the nervous system, in 219 persons, who had come into contact with carbon bisulphide, and his comparative statistics show that these affections are much more prevalent in the rubber industry than in the textile industries.

The Leipsic Psychiatric Clinic[7] reported 50 cases of nervous disorders caused by carbon bisulphide poisoning, of which nearly one-third developed insanity.

The present tendency is to replace carbon bisulphide by the employment of chloride of sulphur in benzine, which is at least a less dangerous agent. As a substitute for benzine, dichlorethane and carbon tetrachloride have been urged. Acetone, turpentine and ether are also employed as solvents instead of CS_2, benzine and benzene.[8]

The Ohio Survey which covered the "cold cure" process in 15 establishments with about 200 employees revealed exposure to benzine, benzol, wood alcohol, carbon bisulphide, carbon tetrachloride, and sulphur chloride, soapstone and sulphur dust. A number of cases of poisoning were diagnosed and quite a number of pale and anæmic persons of both sexes were seen. Inflammatory affections of the skin, cases of dyspepsia and neurasthenia were frequent observations.

Von Harmsen[9] reports that of 220 German vulcanizers, 18.6 per cent. suffered from gastro-enteric catarrh, 11.8 per cent. from tonsillitis and catarrh of the pharynx, 6.8 per cent. from anæmia and 0.9 per cent. from diseases of the nervous system. The statistics of the Sick Benefit Society of Leipsic show a very high morbidity rate in the rubber industry, viz., 1028 days per 100 workers per annum, and a mortality hazard of 0.90 per cent. The same statistics give the percentage of carbon disulphide poisoning as 1.83, including a considerable number of abortions and premature births, which were attributed to mild forms of acute poisoning.

10. **Rubber Cement Manufacture.**—In this process, the powdered rubber is dissolved by means of benzine, benzol or carbon bisulphide, either singly or combined and sometimes other ingredients are added. The mixing is frequently done by hand labor in open containers, but in modern establishments the process is carried on mechanically and the poisonous solutions and fumes are confined within rotary mixers, and the rooms are supplied with efficient exhaust ventilation. The filling of cans or compressible tubes with cement may be done openly or within a confining apparatus. The process involves comparatively few employees but an undue exposure to the industrial poisons already mentioned. The degree of danger varies with the mechanical protective measures provided and the care exercised by the individual worker. There is also considerable danger from fire and explosions, on account of the inflammable character of the vapors present; hence isolated, unheated out-buildings are often used.

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11. **Rubber Reclaiming.**—This is a special industry for the purpose of reclaiming rubber from waste products and old rubber goods. The process consists in grinding, milling, chemical treatments and drying, and the product is generally known as "rubber shoddy." In addition to the rubber solvents already mentioned, mineral acids and alkalis are used. Rambousek[10] cites cases of aniline poisoning, where aniline was used for the extraction of rubber. The grinding operation involves exposure to dust of a mixed character, often containing dangerous amounts of lead. Some of the processes involve exposure to a very steamy atmosphere and wet sloppy work. During the grinding process, which is done between steel rollers, the material becomes heated and emits bluish and very irritating vapors, probably antimony.

Dr. Crzellitzer, cited by Neisser,[11] reports a case of retro-bulbar neuritis in a man engaged in grinding refuse and fragments of hard rubber. On the second day of his work the patient developed symptoms of headache, nausea, dizziness and inflammation of the eyes. Dr. Crzellitzer leaves the question open, whether during the grinding process sufficient heat was developed to disengage carbon bisulphide, or whether we have to deal with other low ebullition compounds, as the cause of these symptoms.

12. **Rubber Substitutes.**—Substitutes for rubber have been made by boiling a mixture of vegetable oils, sulphur, and resin, with chloride of sulphur, during which process sulphureted hydrogen is evolved and may be inhaled.

Preventive Measures.—In view of the far reaching and evil consequences of bisulphide of carbon, especially in the rubber industry, its use should be prohibited. Until this is accomplished, the curing vats should be low placed since the vapors are heavy, and local confining apparatus and copious downward exhaust ventilation for the removal of all toxic vapors should be provided. The practice of dipping with unprotected hands should be stopped. Special curing and drying chambers with exhaust ventilation should be provided and all vessels containing poisonous agents should be kept covered. Medical inspection and suitable work suits and facilities for bathing should be provided, and all dangerous processes should be limited to 2½-hour shifts, with alternation of work.

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SECTION IV

PROCESSES INVOLVING EXPOSURE TO LEAD POISONING

BY GEORGE M. KOBER, M. D., Washington, D. C.

Lead Smelting. Manufacture of White Lead. Lead Pipe and Plumbers' Supplies. Plumbers, Gas and Steam Fitters. Manufacture of Shot. Painters. Sand-papering of Lead-painted Surfaces. Preventive Measures. (See also Section V, Division I, Part I, Lead Poisoning in U. S. and III on India-rubber Industry, and Division II, Chap. IV, on The Printing and Publishing Industry.)

WORKERS IN LEAD AND ITS COMPOUNDS

All occupations involving the handling of lead and its compounds and in which lead fumes or particles of lead dust may be inhaled, swallowed, or absorbed must be regarded as dangerous to health. Layet has enumerated 110 trades in which lead poisoning is liable to occur, but there are doubtless many other occupations in which lead intoxication may take place. Neisser,[1] for example, reports a fatal case in a man who spent most of his time in fishing and contracted lead poisoning by the habit of placing the lead sinker of the fish line in his mouth; another fatal case occurred in a saddler, who did not handle lead in any other way except that he used occasionally a small block of lead in pounding leather.

Great Britain has the most complete statistics on the subject of lead poisoning, notification of which was made compulsory in 1895. Dr. Legge[2] has tabulated 6762 cases with 245 deaths, which occurred in different industries of that country between 1900 and 1909, both inclusive. I have arranged his table according to frequency as follows: Of the 6762 cases, 1295 were reported in the white lead industry, 1065 in the pottery industry, 697 in coach building, 422 in the manufacture of paints and colors, 452 in paints used in other industries, 412 in smelting of metals, 285 in electric accumulator works, 269 in ship building, 217 in plumbing and soldering when carried on in factory premises, 211 in file cutting, 200 in printing, 138 in tinning and enameling, 109 in the manufacture of sheet lead and piping, 108 in red lead works, 75 in brass works, 52 in enameling iron plates, 48 in glass cutting and polishing, 48 in litho-transfers and 659 in other industries. These figures do not include cases among house painters and house plumbers.

Dr. E. R. Hayhurst[3] reports from the State of Ohio between July, 1913, and November 30, 1914, 514 positive and 138 tentative cases of lead poisoning in 48 industries, chiefly in the manufacture of electric apparatus, storage

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batteries, Babbitting metals, solder, painting automobiles and carriages, and in the pottery and rubber industry.

Wächter,[4] based upon a study of 1383 cases of lead poisoning in Prussia, rates the risks as follows: white lead workers, 33 per cent.; red lead workers, 32 per cent.; shot and pipe workers, 20 per cent.; painters, 7–10 per cent.; lead and zinc smelters, 8–9 per cent.; printers, 0.5 per cent.

Mining of lead ore involves the general hazards incident to metalliferous mines. The danger from lead in this operation is generally regarded as slight, unless the ore contains lead in the form of carbonate or sulphide. It is not improbable, however, that the subtle forms of lead poisoning may occur in all lead-ore mining from the ingestion of lead dust.

Lead Smelting.—Lead is mostly extracted from galena, which is a native lead sulphide, by different processes, known as the roast and reaction process, the precipitation process and the roast and reduction process. The latter is most commonly employed. Lead usually contains also other metals, such as silver, copper, arsenic, antimony, iron, zinc, bismuth and tin. For the recovery of silver the lead is melted and oxidized in a cupola furnace, during which process the lead is converted into a red oxide, litharge, and the metallic silver remains behind.

The English statistics of 412 cases of lead poisoning in smelting of metals include 253 cases in persons engaged in the smelting of lead ores and in desilvering lead, 99 in the manufacture of zinc and 60 cases in smelting other lead-containing metals, such as iron or copper ore.

The men who charge and attend to the furnaces, the flue cleaners, and those employed at the lead and slag runs, and in the crushing and packing of litharge, are most exposed to lead fumes and dust.

The operations at the refining furnaces and the crushing and mixing and transport of the ore are less dangerous.

Of 999 employees in Prussian lead smelters[5] in 1905, 177 suffered from lead colic or lead palsy, involving 3056 days loss of work. In recent years conditions are more favorable. In 1909 among 550 lead smelters near Hildesheim, only four cases of lead poisoning were reported and at Potsdam only five among 660 workers.[6]

Müller[7] found from 0.0029 to 0.00569 gram of lead per cubic meter in the air of smelting works, and the water in which the hands of litharge grinders and sifters were washed was found to contain 1.27 grams of lead per liter.

White Lead.—The manufacture of white lead has been described by Dr. Alice Hamilton on page 106, and it may be well to emphasize the fact that the men who empty the stacks or chambers, and those who are engaged in washing, crushing, grinding, sifting and packing of dry white lead are especially liable to plumbism. In Massachusetts[8] we are informed that the men who empty the stacks do not remain long at work, partly because of their roving disposition and the disagreeable character of the work; others acquire lead poisoning and are obliged to quit, and even those of good intentions

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rarely work more than a month. According to Prof. Roth,[9] quite a number of German proprietors of white lead works purposely employ men of roving disposition or make frequent changes, quite unmindful of the fact that this very class, because of ignorance and neglect, are much more liable to contract lead poisoning. The factory inspectors of Great Britain long ago pointed out the danger of employing casual labor. Dr. Legge reports that of 1463 persons employed in white lead works, the incidence of lead poisoning was 6 per cent. among the regular employees, against 39 per cent. in the casual workers.

Preventive Measures.—Sanitarians have urged for years the substitution of less harmful substances for lead, such as zinc oxide and lithopone for lead paints, leadless glaze in potteries for lead glaze, tin blocks for lead beds in file cutting, etc. Excellent preventive measures have been promulgated and as a result of factory sanitation, official regulations, and educational methods a gratifying reduction in the morbidity and mortality from plumbism has taken place. For example, in Great Britain, where compulsory notification exists, the number of cases of lead poisoning has been reduced from 1278 in 1898 to 505 in 1910. The percentage of severe cases in men was 23.9 against 13.9 in females, possibly due to the fact that females have cleaner habits and also stop work more promptly upon the appearance of the first symptoms.

The general principles embodied in official regulations should provide for (1) the removal of the toxic fumes by forced ventilation; (2) reduction of dust production by the wet processes and automatic machinery, and its removal by exhaust ventilation. (3) Respirators should be worn and the hours of labor for those engaged in the oxidation chambers and other dusty processes should be shortened, with alternation of work. (4) Lockers should be provided so as to protect the street clothing worn by the employees. (5) Washing and bathing facilities should be provided. (6) Special lunch rooms are necessary. (7) Semi-monthly medical inspection of employees, suspension of work in case of sickness, and re-examination before returning to work should be enforced. (8) Alcohol and tobacco should be forbidden during the working hours.

In the way of individual hygiene, personal cleanliness, temperate habits, good food and fresh air are important. The work should be done in overalls, frequently changed and washed. The head should be covered with suitable caps; short hair and no beard offer distinct advantages. Before meals the exposed parts of the body should be "dry dusted," the hands, face and nostrils thoroughly washed with soap and warm water, and the mouth and throat rinsed with a solution of tartrate of ammonia.

All health rules should be conspicuously posted and the attending physicians should educate the employees so that they may guard against the dangers, remembering always Sir Thomas Oliver's view that lead poisoning may be easily caused and almost as easily prevented, especially by personal cleanliness. The so-called sanitary drinks, made of epsom salt,

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lemon juice, etc., are of little avail; there is at present no real antidote to this subtle poison.

Lead Pipe and Plumbers' Supplies.—The men engaged in the manufacture of lead pipe and plumbers' supplies are exposed to lead poisoning not only from the fumes, but also from dust in handling sheet lead.

The manufacture of solder involves similar risks. Common solder is a mixture of equal parts of lead and tin. Hard or fine solder is composed of two parts of tin and one of lead, and coarse or soft solder contains two parts of lead and one of tin.

The Massachusetts Report[10] speaks approvingly of the efforts of the manufacturers of plumbers' supplies to carry off the dust and fumes by hoods and exhaust ventilation, that all the employees took the necessary precaution and in no instance was it possible to trace a case of lead poisoning to faulty methods. In an establishment for the manufacture of solder, although rats, cats and dogs appear to succumb to lead poisoning, only one case of plumbism occurred among the employees in 35 years.

Plumbers, Gas and Steam Fitters.—These occupations are often carried on under unfavorable environments, especially in new construction, which involves ditch work and exposure to dampness in cellars and basements. Exposure to lead is unavoidable in handling, cutting, and filing of lead pipes, bends, traps, etc., and also in the employment of white or red lead for making joint connections. The use of solder involves exposure to lead and acid fumes and the products of combustion of either an open charcoal fire or a gasoline torch. Cases of arseniureted hydrogen poisoning have been reported in plumbers, and the hydrogen flame constitutes a distinct menace. Unwashed hands doubtless play a frequent rôle in the ingestion of lead. Sternberg[11] has reported wholesale cases of plumbism in Vienna among this class of workers. The accident risk in new construction work is not inconsiderable; the morbidity hazard among the Leipsic plumbers and gas fitters amounts to 836 days per 100 workers per annum, with a mortality hazard of 0.60 per cent. Hoffman's[13] statistics, based upon 1133 deaths among plumbers, show that 32.9 per cent. of the mortality from all causes was due to tuberculosis, followed by accidents with 11.7 per cent.; diseases of the urinary organs, 11.1 per cent.; pneumonia, 7.7 per cent.; heart disease, 5.2 per cent.

Manufacture of Shot.—In the manufacture of shot, metallic lead is fused with arsenious acid or more frequently with metallic arsenic in suitable proportions. Arsenic is added to harden the shot. When the mixture is ready it is poured through sieves of different sizes at the top of a tower filled with water at the base, which hardens the lead drops. The shot is subsequently polished by means of graphite in a revolving drum. The fusion process involves exposure to both lead and arsenic unless guarded against by suitable crucibles.

Painters.—This occupation covers a large number of specialties, and

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some industrial statistics include even glazers, whitewashers and paperhangers under the general head of painters, decorators and paperhangers.

The general classification suggested by Dr. Hamilton[12] includes house, sign, and ship painters; coach painters, inclusive of wagons and carriages, automobiles, railway and street railway coaches, bridge, tank, and structural iron painters; painters of agricultural implements, furniture, picture frames and mouldings. It is obvious that the ocupational risks vary considerably in the different departments, and depend to a great extent upon the materials employed, the character of the work, the conditions under which the work is performed and the precautions taken by the workers.

Hoffman's Industrial Insurance Statistics[13] show that of 2783 deaths among painters 23.8 per cent. were caused by tuberculosis, and 9.9 per cent. from other respiratory diseases, making a combined rate of 33.7 per cent. from diseases of the respiratory organs; the rate from lead poisoning was 1.5 per cent.; from nervous diseases 10.7 per cent.; from heart, kidney and liver diseases 25.9 per cent. The German figures, cited by Fleck,[14] are 41.6 per cent., 1.3 per cent., 7.8 per cent. and 20.8 per cent. respectively. The Leipsic Sick Benefit records show 1017 days of sickness a year per 100 members and a mortality hazard of 0.72 per cent.

The composition of paint may be very simple or quite complex. Among the poisonous pigments should be mentioned those containing arsenic, lead and mercurial compounds, and among the liquid vehicles, driers and varnishes, which may act as industrial poisons, are turpentine, benzine, benzol, creasote, wood alcohol, asphaltum, amyl acetate, banana oil (a mixture of amyl acetate, acetone and benzine) and carbon disulphide.

All quick-drying flat-finish paints or varnishes are generally made of leadless pigments, but usually contain some of the petroleum products, which may be a source of benzine poisoning, as the work has to be done in closed rooms because open windows and drafts cause the paint to dry too rapidly and render the coat streaky.

The danger from arsenical and mercurial pigments has been pointed out no pages 3, 126, 486. Lead poisoning among painters has long since characterized this occupation as a dangerous trade. Koelsch[15] in a careful study of 5000 painters in Munich found that only 8 per cent. were over 50 years of age; the majority were between 16 and 40 years of age. Of this number 13.83 per cent. had experienced symptoms of lead poisoning; about one-third had suffered from more than one acute attack. Colic occurred in about 85 per cent. of the cases; lead palsy in 3.5 per cent.; joint affections in 2.7 per cent.; sciatica, visual disturbances, renal involvement and gout in about 2 per cent. of the cases. Cases of lead poisoning involving the brain were not encountered, since the physical examination was limited to actual workers; the hospital records of Munich show, however, a proportion of 5.5 per cent. About 29 per cent. of all the workers examined showed objective or subjective symptoms of lead poisoning; 11.8 per cent. revealed the characteristic gum

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lines; 6.5 per cent. were anæmic; digestive disorders, headache, tremor, dizziness, circulatory and kidney disorders and rheumatic affections were not infrequent.

Koelsch's general percentage corresponds very closely with the results obtained by the Illinois Commission on Occupational Diseases for 1911, which show that of 578 cases of lead poisoning, 27 per cent. were painters. The statistics of Pratt and Andrews, cited by Dr. Hamilton,[12] indicate a higher percentage. The average age of 1388 painters, decorators and paperhangers in Chicago in 1911 was 41 years and 3 months. It is of interest to note that the painters examined by Dr. Hayhurst were encouraged to state what they considered the most unhealthful feature of their work and 99 out of 100 mentioned the lead-paint dust from sand-papering, 70 complained of benzine, 64 of turpentine when used in close quarters, and nearly all stated that the fumes of benzine and turpentine in hard oiling made them sick temporarily. Three of the men had been victims of wood alcohol blindness, contracted while working in brewery vats.

Hayhurst[8] in his Ohio Survey reports 154 cases of lead poisoning, three from benzine and naphtha, two from turpentine and one each from varnish and wood alcohol.

It has been shown by Gardner[16] that drying paints emit no metallic vapors, but they may evolve benzol, formic acid, aldehyde substances, carbon dioxide and carbon monoxide, and while all are more or less harmful, he very properly maintains that carbon monoxide may be a fruitful cause of anæmia among painters. These conclusions naturally suggest the need of copious ventilation.

Causes of Lead Poisoning among Painters

1. *Mixing Dry Lead Pigments with Oil or Paints.*—This source of danger is not pronounced, as most of the paints are manufactured in special establishments and very few painters have occasion to use dry pigments, except those employed in structural iron painting who use red lead. Of 100 cases of lead poisoning cited by Dr. Hamilton[12] only two had used dry white or red lead. Unless the mixing is done carefully, or in inclosed machinery, there is danger from dust inhalation.

2. *Sandpapering of Lead Painted Surfaces.*—This is universally acknowledged to be the most important cause of lead poisoning among painters, as the evolution of dust by dry sandpapering or rubbing with pumice-stone, naturally favors inhalation or ingestion of dust containing lead or other toxic agents. It is believed also that the sharp, angular character of sand or pumice-stone dust renders the mucous membrane vulnerable and hence more susceptible to the absorption of toxic dust.

The removal of old paint by means of benzine, wood alcohol, compressed air, chipping, or scraping is equally dangerous work, especially when this has to be done in confined spaces, notably between the outer and inner shell

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of steel ships. Burning off the old paint by means of a small gasoline torch is a safer method, as there is little or no danger from lead fumes, unless the workman permits the flame to play long enough on the surface to volatilize the lead and produce a decided smoke, in which case particles of lead may, in the opinion of Prof. Stieglitz, be carried off mechanically with the smoke. The inhalation of lead dust from dirty drop cloths and overalls is doubtless a frequent cause of plumbism among house painters and decorators. And for obvious reasons the men should also be careful not to expose their street clothing to lead-containing dust.

The ingestion of lead through unwashed hands, etc., while eating, drinking, smoking, or handling tobacco and pipes, is naturally a frequent cause of plumbism. While every painter knows that "hot water and soap and time enough to use them" are important protective measures, the fact is that these facilities, except in shop work, are not always available. In new construction work and vacant houses the water may not be turned on, and the workman is obliged therefore to clean his hands with benzine, turpentine, etc., provided for cleaning the paint brushes, and those who fear that these agents drive the lead into the pores of the skin, simply resort to a piece of rag, cotton waste, or paper for the removal of gross particles of lead.

Among the contributory factors to lead poisoning should be mentioned chronic alcoholism. My personal observations lead me to conclude that the alcohol habit is quite prevalent among painters, and old topers do not hesitate to tipple alcohol from shellac and even denatured alcohol. Pieraccini has pointed out that lead in the system creates a desire for alcohol, and that the combined effects quickly damage the kidneys and arteries. The life Insurance statistics, on the other hand, indicate that only 1.4 per cent. of the 2783 deaths among painters were caused by alcoholism, as against 1.9 per cent. for plumbers and masons and an average of 1.5 per cent. for 103,434 occupied males. The German and French statistics are not so favorable.

The liability to accidents, such as falls from ladders and scaffolds, is not inconsiderable and house painters are likewise exposed to electric shocks from overhead wires. Exposure to the elements doubtless plays an important rôle in the causation of catarrhal and rheumatic affections and diseases of the respiratory organs. Pneumonia, according to Hirt, is more common among house painters than in decorators and varnishers. Diabetes is reported by Rosenfeld as not uncommon. Affections of the eyes, from exposure to dust, toxic fumes, etc., are likewise quite frequent, and dental and gastric diseases are equally common. See also diseases of the circulatory organs, arteriosclerosis and diseases of the kidneys. Flat-foot, knock-knee, and varicose veins as a result of prolonged standing are not uncommon, and housemaid's knee has been noted among men, who make a specialty of painting or polishing floors. Callosities of the palm of the hands, and inflammatory affections of the skin of the hands and arms caused by turpentine and petroleum products are not infrequent.

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Preventive Measures.—Dr. Hamilton has pointed out on page 116 the salient features in the prevention of lead poisoning among painters. Professor Sommerfeld regards dry sandpapering as the most fruitful cause of lead poisoning and hence no effort should be spared to substitute moist processes and to resort to all the precautions referred to on page 514.

The French Government has adopted radical measures for the suppression of white lead paint, to take effect January 1, 1915. According to the Monthly Review of the U. S. Bureau of Labor Statistics for November, 1915, a committee appointed by the Home Department of Great Britain recommends the enactment of a law prohibiting the importation, sale, or use of any paint material containing more than 5 per cent. of its dry weight of a soluble lead compound. Germany has forbidden the mixing of white lead by hand, and insists that the rubbing of fresh or old paint with sandpaper or pumice may be done only after previous dampening. Austria has a similar law and also forbids the use of white lead for inside work except in certain cases. The German law also requires that the contractor for house painting shall provide caps and suitable overalls, kept in order and washed, and temporary working sheds, where the men may keep their street clothing, keep and eat their lunches, supplied with warm water, soap, wash-basins, nailbrushes, and towels. The employer must suspend from work any painter known to him to be suffering from lead poisoning. The law regulating painting in factories also forbids the use of alcohol and tobacco, and provides for medical inspection of employees at least every 6 months. A few of the states have enacted laws for the protection of workers in lead, some of the laws cover also painters when employed in factories or workshops.

The Brotherhood of Chicago painters and paperhangers during a strike in April, 1913, formulated the following reasonable demand: "*no* workman or apprentice shall be required to use any poisonous substance or material injurious to health, such as wood alcohol, varnish remover, oxalic acid, or the sanding of lead, etc., unless they are protected with respirators, gloves, etc., same to be furnished by the employer; nor shall they be required to use any dirty or insanitary waste, rags or drop-cloths. There shall be an allowance of 5 minutes for wash time in each 4 hour's work, and where lead or other poisonous material is used, the employer shall furnish hot water, soap and towels to the workmen. The officers and members of the organization shall enforce this clause."

Robinson and Wilson[17] inspected five factories engaged in the manufacture of *white lead and paints*, and examined 226 workers, none of whom were found to be tuberculous and but five whose physical condition was below par. This favorable showing is attributed to the strict medical supervision over the workers, primarily intended for the prevention of lead poisoning. In operations producing dust this hazard was minimized by the use of blowers.

Only one of the establishments had any cuspidors but in insufficient numbers. Spitting on the floor was noted in all. Toilet and washing facili-

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ties were good in three and only fair in three establishments. In the white lead factory the floors were mopped every morning, while two of the other places were sprinkled and swept in the morning and two others were swept after working hours.

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SECTION V

PROCESSES INVOLVING EXPOSURE TO MERCURIAL POISONING

BY LUDWIG TELEKY, M. D., Vienna, Austria, AND GEORGE M. KOBER, M. D., Washington, D. C.

Mining.† Smelting.* Manufacture of Physical Apparatus.* Extraction of Gold and Silver.* Fire Gilding.* Manufacture of Electrodes and Storage Batteries.* Mercurial Chemical Compounds.† Mirror Plating.† Hatters' Fur Cutting and the Felt Hat Industry.† General Preventive Measures.†

WORKERS IN MERCURY AND ITS SALTS

Mining of the Ore.†—Of the total output of 4300 metric tons in 1912, 855 tons were produced in the U. S. Mercury is chiefly obtained from the sulphide of mercury ore, commonly known as cinnabar (HgS). Pure cinnabar contains 86.2 per cent. of mercury, the ordinary ore contains generally less than 1 per cent., exceptionally 2.5 per cent. as in Brewster County Texas, and as high as 9 per cent. in the mines of Almaden in Spain. Since cinnabar is insoluble there should be no danger from this source. Dr. Pope of London as early as 1665 referred to mercurial tremor of the hands among the miners of Italy. The eleventh census of the United States, page 28, reports that 10.44 per cent. of the men employed in the New Almaden mines suffered from mercurialism. Such instances are doubtless due to the fact that mercury in certain mines and galleries has been known to collect in small and large drops, which under the influence of a very warm and moist atmosphere is volatilized and inhaled.

According to Dr. Teleky the mines at Monte Amiata are quite free from such a risk, while those of Idria and especially the Spanish mines present this danger. In all such cases it has been found practicable to reduce the danger by copious ventilation, and by a change of crews to other locations, so that the men may not work over 4 hours in dangerous places.

Smelting.*—Mercury is usually obtained by subjecting cinnabar (mercuric sulphide) to a roasting process in furnaces. By this procedure the sulphur combines with oxygen, forming sulphur dioxide, while the mercury which distils off, is collected in special receptacles. The aim of the technologist on the one hand is to bring about a comparatively complete disintegration of the ore and on the other he seeks to keep the escape of mercurial vapors from the furnaces down to a minimum. In this endeavor technology and hygiene join hands, because mercurial vapors are a source of

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grave danger to the workmen. Above all things in this industry it is necessary to build condensers of sufficient spaciousness so as to make the condensation of mercury as complete as possible. At Idria, in Austria-Hungary, the outlet of the stack is 158 meters above the arch of the smelter and the total content of the condensing plant is 7631 cubic meters.

If a condensing plant is not sufficiently effective there occurs on the one hand a colossal loss of mercury, and on the other there is danger of jeopardizing not only the health of the workmen but also that of persons living in the vicinity of the smelter.

In Idria, in previous years it was apparently a common thing to have stock poisoned as a result of grazing on lands in the neighborhood of the smelter.

Even when the amount of mercury escaping from a stack is small it must be regarded as dangerous to smelter workers when the outlet of the chimney is not sufficiently distant from the smelter or if it is badly placed.

The greatest source of danger, however, is from vapors escaping from the furnaces within the smelter. Escape of vapors can be prevented only when a negative pressure exists in the furnaces. A negative pressure is maintained by a mechanically driven ventilator (fan) which exerts its activity throughout the entire furnance system. It often occurs that the wings of a ventilator become corroded by the acid vapors which pass through the stack and consequently it is important at all times to hold an extra fan in reserve.

In shaft furnaces, employed in the working up of coarse ore, difficulty is experienced in affecting a complete seal for preventing the escape of mercury vapors. Giglioli, for instance, found that the apparently efficient seal of Spirek also allows mercury vapor to escape. A condition similar to the one prevailing in the shaft ovens often obtains with the continuous automatic reverberatory furnaces (Fortschaufleröfen).

In the most modern furnaces (Czermak-Spirek Schüttrostöfen) the fine ore which is worked up therein serves as a seal. Even with this device one must be cautious. In working with this type of oven it is especially necessary to feed only ore which has the proper degree of fineness, and to have the uppermost layer of ore of a proper height. It is, furthermore, important to have a pipe with an especially strong suction in the upper layers of ore for the purpose of intensifying an existing negative pressure in the furnace.

The greatest advantage, from a hygienic standpoint, obtained by employing the Czermak-Spirek (Schüttrostöfen) furnace, is in the fact that the ore under treatment falls by gravity during the smelting process and consequently does not require further handling by the workmen.

With all types of furnaces, however, great care must be exercised when removing the spent ore. In this case it is especially necessary to see that the workman who opens the grate bars is not overcome by mercury vapors and dust. This can be prevented by placing a wall of sheet-iron between the spent

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ore cart and the spot where the workman usually stands, and by installing suction pipes in appropriate localities. The carts, when filled with spent ore, which is cooled, should be conveyed to the spent ore heap automatically. (At Idria spent ore contains 0.00036 to 0.00148 per cent. of Hg.)

The gases which are produced in the furnace by the smelting process go over into condensers of large dimensions. In the condensers a soot technically called "stupp" collects which consists of a mixture of finely divided mercury, unburned particles of carbon, undecomposed hydrocarbons, ore dust and ash. As only a comparatively small portion of metallic mercury (5 to 10 per cent.) is obtained from the condensers, it becomes necessary to subject the soot (stupp) to special treatment in order to extract the mercury which it contains. The mercury content in stupp is variable, the amount depending upon the locality from which it is obtained.

The removal of soot from the condensers must be regarded as an occupation which is quite detrimental to the health of those engaged in it. When practicable plenty of water should be played during the operation. Especially dangerous is the operation in cleansing the entire canal system. At this time it is necessary to stop all smelting operations, and once each year at Idria the works are at a standstill for this purpose. To prevent disease as much as possible from this source requires the wearing of special clothing, the employment of respiratory apparatus and baths at the end of each working period. The working period should not be over 4 hours for each shift. The extraction of mercury from stupp (soot) is done with specially designed presses. The presses should be provided with strong suction devices for the purpose of carrying off noxious dusts and gases.

Every manipulation associated with the handling of stupp should be regarded as a source of danger to health because poisoning can occur from volatized mercury or by the absorption of finely divided mercury in a manner similar to the poisoning which sometimes occurs when blue ointment is rubbed on the skin. There is even danger of poisoning, through volatilization and absorption, during the operation of weighing and filling mercury into cast-iron flasks, etc. Accordingly it is advisable to keep the flask covered and to avoid spilling the metal, and to prevent mercury from coming into contact with the hands. In rooms where metallic mercury is handled the floors should be smooth and tight and should slope in the direction of a vat for the collection of mercury droppings. The same requirements hold good for work tables on which mercury is handled.

Eating should not be permitted in the smelters and as additional general precautionary measures the help should be required to wash the hands and to take a bath daily before leaving the smelter. It is also important that the help wear clothing made of close-meshed fabrics for the purpose of preventing the penetration of the underwear with soot and mercurial vapors. Where this precaution is not taken the workmen are apt to carry mercury into the home. In doing this the workman jeopardizes not only his own health, by

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being continuously in an environment of mercury, but also that of the other occupants of the same house. At Idria cases of poisoning are often noted among members of a mercury worker's family. This is especially true where children sleep in the same bed with a father who is engaged in a mercury works.

From what has been said it is evident that the untoward effects of mercury can be minimized if effective technical and hygienic appliances are installed in the smelter. It is also obvious that a complete prevention of disease from this cause is impossible.

Under these conditions much can be gained by making frequent changes among the help and no person should be allowed to work over 4 weeks at a time in the smelter. Each period of smelter work should be followed by a comparatively longer period of labor at a less dangerous task, as mine work, etc. At Idria the help is required to work 1 month in the smelter and the following 2 to 3 months in the mine.

Although the technical equipment at Abbadia San Salvador is not less perfect than that of the works at Idria, the incidence of mercury poisoning is much greater. At Idria among 1200 miners I found nine severe cases of mercury poisoning. Five of these cases had received pensions for several years, and the remainder were still engaged in active work. I have frequently observed slight cases of mercurialism and among a group of 259 woikers there were only 19 who gave a history which would rule out mercury poisoning. The death rate at Idria is abnormally high and many die from tuberculosis.

Manufacture of Physical Apparatus.*—The utilization of mercury for constructing various kinds of physical apparatus was and still is a source of mercury poisoning.

In constructing the mercury barometer a great deal of mercury is employed and it often happens that globules of mercury fall to the floor of the factory and become a source of danger to the operatives. Poisoning from this source is not as frequent as formerly because the aneroid barometer has displaced the mercury instrument to quite a large extent.

Mercury is also utilized to a considerable extent in the manufacture of thermometers. This occupation, which is still carried on in many cases in dwellings, is especially dangerous because it is necessary to heat the mercury in order to expel air from the thermometer tubes. In order to prevent poisoning it is essential to keep the living quarters distinctly separate from the workshop.

The tables in the workshop should be bordered with raised strips to prevent mercury from falling to the floor, and the table board should incline in the direction of a mercury-catching receptacle.

In the manufacture of incandescent lamps vacuum pumps are much used. The Sprengel and similar air pumps, whose powers depend upon the

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fall of a small stream of mercury through a narrow bore tube, are still frequently used. The glass parts of the pumps, when broken, often become a source of danger—due to spillage of mercury—to the working force, which is composed chiefly of women. It is therefore important to set up the pumps on tables similar to the ones described in the section on the thermometer, to prevent mercury coming on the floor and furthermore to replace as quickly as possible all cracked parts of pumps. The Sprengel pump in Germany and America is gradually being displaced by the rotary pump, but this too requires mercury for its makeup. Although the danger of poisoning from breakage of these pumps is not as great as with the Sprengel pump, poisoning can occur when the apparatus is cleaned. In the rotary pump there is a tendency for the mercury to collect in the joints and cavities and on the glass parts, but in the latter case it is due to impurities in the mercury. The mercury of pumps when impure must be cleansed by a process of distillation or by shaking it with sulphuric or nitric acid. In the first method when the distilling apparatus is imperfect and allows the escape of vapors the health of the operator is endangered. In the second method the acid is removed from the metal by washing with water. The water remaining incorporated with the metal is removed by heating, in consequence of which some of the mercury is rendered volatile. Here too is a source of poisoning.

Mercury pumps are also used for exhausting Röntgen (X-ray) tubes. In this work the quantity of mercury employed is much greater than is used for exhausting incandescent lamps, and the danger arising from spillage of mercury and breaking of pumps is still greater. I have seen the old Toepler pump employed for this purpose.

Inasmuch as workmen engaged in the repair and cleansing of mercury pumps are also exposed to the danger of poisoning, it is obvious that a periodic change of hands is indicated. It seems highly desirable from a hygienic standpoint to dispense with the use of the mercury pump and to employ instead Geryk pumps, such as have been introduced into Germany, America and elsewhere.

There are also other physical apparatus in which mercury is used. Notably among these are instruments for taking electrical measurements. English industrial inspectors have reported cases of poisoning in those who use these instruments.

I have also found numerous cases of poisoning among the help engaged in making a new kind of press gas lamp. By an ingeniously regulated volatilization and recondensation of the mercury the pressure of the illuminating gas is increased in the lamps.

Of 64 cases of mercurialism reported in Great Britain, 17 occurred among workers in electric meters and 16 were engaged in the manufacture of philosophical instruments (G. M. K.).

In this connection it may be stated that Edsall[1] relates the case of two dentists who contracted mercurial poisoning from the habit of working up the

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amalgam in the palms of their hands. He also refers to a patient engaged in working with physical apparatus and who presented symptoms of excessive irritability and apprehensiveness, also persistent diarrhea. Edsall[1] traced the source of poison to the fact that the patient kept an open jar of mercury on his office desk, and frequently spilled quicksilver on the floor of his office and bed room without paying any attention to it. After removal of the cause the symptoms disappeared. (G. M. K.)

The Leipsic morbidity rate for makers of instruments of precision is 955 days per annum per 100 members, and the mortality hazard 0.78 per cent. (G. M. K.).

Extraction of Gold and Silver.*—The property of mercury to form an amalgam with metals, especially the precious metals, is made use of in the extraction of gold and silver. In Europe, however, mercury is no longer employed for extracting silver, but the process is apparently still utilized to a considerable extent in America.

In Mexico the Patio or heap amalgamation process is still in use to a limited extent. In this procedure the finely reduced silver ore, placed in large flat piles, is strewn with sodium chloride, roasted chalcoyprite and mercury, and mixed with a shovel. The heaps are then treated by a burro or kneaded with electrically driven ploughs. After several weeks of this kind of treatment the heavy amalgam is treated in agitators and settling tubs.

Another method of extracting silver is the Washoe or pan amalgamation process. Here moist ore is stamped to powder and transferred to cylindrical or slightly conical steam-heated fans provided with a device termed a muller for stirring or rubbing the ore with mercury, copper sulphate and common salt. The amalgam is then separated from the residue in a settler provided with an agitator.

In another process the ore before amalgamation is converted into chloride by roasting it with salt in a reverberatory furnace. The silver chloride so produced is then reduced with metallic iron or copper and the silver is then amalgamated by the Reese-River pan amalgamation process.

In all of the processes discussed the silver must finally be obtained from an amalgam. This is done by diluting the amalgam with mercury and filtering the diluted amalgam through canvas. This results in the retention of an amalgam in the canvas cloth, which consists of one part of silver to seven parts of mercury. The mercury in the retained amalgam is removed by distillation. During the preparation of amalgam, irrespective of whether it is done in heaps or in flat open pans, a volatilization of mercury occurs. The vapor, as such, is detrimental, as already pointed out, from a hygienic standpoint.

A less dangerous procedure seems to be the water amalgamation process carried on some years ago at Freiburg, Saxony. The loss in mercury, which can by no means be all due to volatilization, in the Patio process is 1.5 kg.

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per kilo of silver; Burrel amalgamation 0.22 kg. per kilo of silver; and in the Reese-River process 0.25 per ton of ore.

Even more dangerous to health is the operation of removing mercury from the amalgam by distillation. This is particularly true when leaky or otherwise inappropriate distilling apparatus are employed.

Gold is to-day still extracted by the amalgamation method. In this procedure mercury is added to the finely reduced ore, or the amalgamation is done in cast-iron mills simultaneously with the stamping (reduction) process. The latter process—stamping mill amalgamation—is the most widely used.

During reduction in which powerful stamping mills are used, water flows constantly over the ore, and mercury is introduced intermittently. The amalgamation which occurs in the stamping troughs is accentuated by amalgamated copper plates. Each day the amalgam is removed from the trough; that adhering to the copper plates is also scraped off and treated in a manner similar to silver amalgam, discussed above.

Among other methods for extracting gold is the cyanide process of McArthur and Forrest. In most cases this process is preceded by a preliminary stamping mill amalgamation.

Amalgamation is not resorted to in Europe for obtaining silver, but it is apparently employed to a wide extent in Mexico.

The fact that gold and silver mines are not usually located near larger cites is responsible for our lack of knowledge in regard to mercury poisoning in this industry. We can, however, conclude from our knowledge obtained in other vocations, and from the fact that a large amount of mercury is used and the way it is handled in gold and silver extraction, that poisoning from this source must occur among the workmen. More information is required in this direction and a meritorious piece of work could be done in this connection by hygienists located near such plants and mines.

Attention should also be directed at this juncture to the fact that there are establishments engaged in extracting gold from the refuse of gold-working plants by an amalgamation process. Here, too, the mercury is recovered by distillation.

Fire Gilding.*—Extensive use of amalgamation is made in fire gilding (plating). In this process gold in small pieces is heated in crucibles with 8, 10 and often with 15 parts of mercury, and stirred with an iron rod until complete amalgamation has taken place. The commodity to be gilded, previously smeared with a solution of mercury in nitric or cyanic acid, is painted with the amalgam and heated in an oven until all of the mercury is volatilized. In the preparation of the amalgam, but more especially in heating the commodity coated with amalgam, much mercury vapor is developed. Accordingly it is advisable in establishments of this kind to install powerful suction fans for carrying off the vapors. In addition to

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taking precautions against mercury vapors it is also necessary to use caution when removing the commodities from the fire.

Mercury when heated becomes more liquid (less viscid) and has a tendency to flow. Under these circumstances it is necessary to wipe the object gilded with a wad of cotton or brush in order to distribute the amalgam more uniformly and to prevent blistering. In the operation the eyes and mouth are often brought into close contact with mercury and its vapors.

In England the smaller commodities subjected (uniform buttons) to amalgamation are heated in closed ovens.

In the occupation of fire gilding (plating) more cases of acute mercury poisoning, salivation, stomatitis, possibly gastro-intestinal manifestations, occur than in any other calling. This is due to imbibing larger quantities of mercury in a short space of time. It is obvious from what has been said that complete prevention of disease from this source is not possible. According to my experience the number of cases is relatively large among those engaged in this work, even though many of the individuals work only a few hours each day or only a few days of every week. Fortunately the fire-gilding industry is on the decline and to-day only a few commodities are plated by the amalgamation fire method. Among the commodities still gilded by this method are church crosses, possibly cupolas, lightning-rod points, naval uniform buttons and articles in which a special resistance to the weather is desired. To-day plating is done principally by the galvanic method.

Manufacture of Electrodes, Storage Batteries,* Etc.—A further use for amalgam is found in the manufacture of electrodes. In many cases the zinc pole (electrode) when kept in the sulphuric acid in the battery jar will corrode. As a preventive against corrosion the electrodes previously cleansed with sulphuric acid are coated (amalgamated) with mercury. In this operation mercury is poured on the plates, or the zinc electrodes are dipped into a solution of mercury in nitric acid or nitrohydrochloric acid. Several cases of mercury poisoning from this source have come under my observation.

It should also be mentioned that the rapid solder employed in storage-battery work also contains mercury. I have also seen poisoning in those who manufactured and made use of such solder.

In manufacturing storage batteries (accumulators) corrosive sublimate is added to the plates in an amount of 30 grams per kilo of metal.

Corrosive sublimate and mercuric nitrate are used for Damascenizing gun barrels.

Poisoning from sealing wax colored with cinnabar is also reported, but to me it seems improbable. During the preparation of fulminate of mercury, which is used for making cartridges, instances of mercury poisoning may occur. Poisoning from this source, however, is by no means confined to the ones engaged in its manufacture, in making cartridges, but it can also

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affect the attendants of shooting galleries when the ventilation is bad and where a large number of shots are fired in rapid succession. Instances of poisoning have also been observed among the shooters.

Mercurial Chemical Compounds.†—The manufacture of calomel, corrosive sublimate, nitrate of mercury, red oxide and sulphide of mercury is attended with more or less danger from volatilization of the metal in the subliming processes and likewise by the inhalation and absorption of mercurial dust. Legge[2] found that of 27 chemical workers 4 had more or less salivation and 10 had symptoms of tremor, anæmia and gastric derangements. He believes that by the employment of wet methods throughout, most of the dangers can be eliminated, especially if the dusty processes, such as mixing and sieving, are done as far as practicable by enclosed machinery.

Mirror Plating.†—Of 7584 sick days among 160 mirror makers at Fürth in 1885, not less than 5463 days loss of work were caused by mercurial poisoning. This was at a time when mercury was poured as an amalgam on a sheet of tin foil and the glass plate placed over it and weighted down so as to squeeze out any excess of mercury. Dr. Heucke[3] informs us that this process has been almost completely replaced by the use of a solution of nitrate of silver containing an alkaline reducing agent. The new process is not wholly free from danger, however, as cases of lead poisoning have occurred from the use of red lead with which the plate is backed. This danger can be obviated by the substitution of a coat of varnish. The use of acetaldehyde in connection with the modern process of silvering mirrors should likewise be avoided.

According to Hayhurst,[4] nitrate of silver and tartaric acid have entirely supplanted the use of mercury in this country. There is a distinct need of better ventilation in most of the establishments, especially for the removal of nitric acid fumes. The temperature of the silvering rooms is often excessively hot and the beveling, buffing and polishing with rouge paste is generally carried on in rather damp places.

Hatters' Fur Cutting.†—In the preliminary treatment of the fur for the manufacture of felt hats the workers are exposed not only to more or less dust, but also to mercurial poisoning. The latter danger is due to the fact that during the "carotting process" the fur side of the pelts is brushed with a solution of nitrate of mercury. The proportion of mercury used varies in different establishments. According to Heucke,[3] 100 kilos of the solution contains about 20 kilos of mercury and the average amount of the metal applied to each skin is bout 1.64 grams. Analysis of the dust and sweepings of establishments revealed the presence of from 0.13 to 1.33 per cent. of mercury chloride. The solution generally employed in this country is made up of 4 carboys of nitric acid, 75 lb. of mercury and 50 of water.

In Europe the solution is usually composed of 1 part of mercury, 4 parts of nitric acid and 20–25 parts of water; in some instances 5–6 parts of the acid

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are used. The men employed in making this solution and those who apply it are exposed not only to mercury, but also to nitrous fumes. One of the earliest manifestations of their combined action is a greenish discoloration with subsequent brittleness of the teeth. Rubber gloves usually suffice to protect the hands from the corrosive effects of the solution and the absorption of mercury. With reasonable precaution the chances of mercurial poisoning are quite slight as compared with the next process.

After the application of mercurial solution the skins are taken to special drying rooms and their removal from there is fraught with some degree of danger, since Heim determined in a cubic meter of air of such rooms about 3 cc. of nitrous fumes and 12.5 mg. of mercury. The subsequent storing of the dried pelts is a dusty process and increases the chances of mercurial poisoning with impregnated dust. The skins are next taken to the brushing machine which loosens the matted hair, and after cutting off the head, feet, etc., they are passed to the cutting machine, fitted with sharp knives, which shaves the fur from the skin. The fur is then assorted by female labor, according to quality, color, and part of the body, and is then packed in 5-lb. bundles. The brushing and cutting are both dusty processes and Heim and Herbert found in the air of the workrooms 65 mg. of dust with 5 mg. of mercury per cubic meter, which sufficiently emphasizes the need of efficient exhaust ventilation. The fur from the parts or trimmings which have been cut off cannot be removed by machines and this shearing is usually done by home hand labor, a most objectionable practice, since Heim and Herbert found in the air of such houses 75 mg. of dust with 42 mg. of mercury per cubic meter. In some countries, notably Belgium, most of the processes are carried on as a home industry. As a matter of fact, about four-fifths of the 11,000 to 12,000 furriers in Belgium are still home workers.

The sad effects of this system have been vividly described by Glibert, who finds that one-third of the women and over one-half of the men employed less than 5 years suffer from mercurial poisoning. This danger can be readily appreciated when it is remembered that a sample of the fur as received from the cutting machine was analyzed in the British Government Laboratory and was found to contain 1.34 per cent. of nitrate of mercury. The mercury, according to Legge,[5] "forms a very insoluble combination with the keratine in the hair, which is not removed in the subsequent processes of felt hat making." In view of the fact that the men who handle the mercurial solution are exposed to the fumes and all others engaged in dusty processes to the inhalation of fine fur impregnated with particles of mercury, it is not surprising that Dr. Legge should have found 20 of the 30 employees in one establishment and 27 out of 81 workers in another afflicted with defective teeth, and 17 with pronounced mercurial tremor. This same author, as well as Tylecote,[6] Teleky and J. H. Lloyd, was among the first to recognize that the danger from mercurial poisoning was not confined to hatters' furriers, but that the majority of cases actually occurred in felt hat makers.

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Mrs. L. W. Bates[7] in 1912 collected 102 cases of mercurial poisoning, occurring chiefly in the felt hat industry in New York City and vicinity; she reports only three cases in makers of incandescent electric lamps, two in chemists and two in makers of cosmetics.

The Felt Hat Industry.†—During the last census year 27,091 persons were employed in this industry in the United States, of whom 25,064 were wage earners. Of the total number 72.2 per cent. were males and 27.8 per cent. females; 531 persons, or 2.1 per cent., were under 16 years of age. This industry begins with the utilization of the 5-lb. packages received from the fur cutters. The fur of different grades is mixed and thoroughly picked and separated in special machines, provided with tearing or teasing studded wire teeth, and although these machines are enclosed and provided with exhaust ventilation, more or less dust escapes. The "blowing machines" are employed to render the fur more fluffy and to deposit it, by specific gravity, into graded bins. This process in spite of enclosed machinery is very dusty and it is believed that most of the cases of mercurial poisoning originate in this department. The subsequent manipulation of forming, hardening, sizing, shellacing, blocking and dyeing are all wet processes. Much of the work is carried on in a hot and moist atmosphere, doubtless containing mercurial vapor. Over 30 per cent. of the mercury cases investigated by Mrs. Bates were contracted by formers, hardeners and sizers. Apart from mercurial skin affections, the plankers and blockers not infrequently suffer from eczema and disfiguration and loosening of the finger nails, caused by the use of hot acidulated water, or "planker's acid." The stovers, who handle the hard felt shapes at a temperature of 180°F. in the drying department, are exposed not only to mercurial vapors, but also in some establishments to the fumes of wood alcohol, or alcohol denatured with pyridin bases, employed in the shellacing process to stiffen the hats.

A serious explosion, resulting in the killing of 14 persons and injuring of 50 others, occurred at Denton in England in 1901, from the ignition of spirit vapor in a drying stove. These dangers could be materially lessened by the use of drying chambers, such as are employed in connection with steam laundries, provided with exhaust ventilation and limiting the number of hats which could be placed at one time into the dryer.

The "finishing or pouncing process" consists in smoothing off the coarse hair from the surface of the hat, during which a great deal of very fine dust impregnated with nitrate of mercury is given off. Apart from mercurial poisoning, Heinzerling and Lewin cited by Schütte[8] have pointed out that there is also danger from *arsenical* poisoning, since the fleshy part of hare and rabbit skins is not infrequently treated with a soap, containing arsenite of potassium or sodium. The liability to injuries, especially with the carotting machine, and to burns and scalds, should be mentioned.

It is very evident that hatters are exposed to a very irritant dust and, as a result, catarrhal conditions of the eyes and upper air passages and

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chronic inflammation of the lungs, and pulmonary tuberculosis, are quite prevalent.

Schüttes' German statistics[8] show a morbidity rate per 1000 hatters as follows: diseases of the respiratory organs 44.1; pulmonary tuberculosis 21.6; diseases of the eye and ear 30; diseases of the skin 17.1; injuries and burns 18.1; industrial poisoning 22.5; diseases of heart and blood-vessels 13.9; diseases of the digestive organs 28; diseases of the nervous system 21.6. Apart from the injurious factors already mentioned the unsatisfactory working conditions and sedentary habits, especially of the female finishers, are doubtless responsible for an excessive amount of sickness.

Hoffman's Industrial Mortality statistics, based upon 832 deaths among hatters, show that 278, or 33.4 per cent., were caused by consumption, 71 by pneumonia, 12 by bronchitis and 14 by other respiratory diseases, making a combined mortality rate of 45.1 per cent. from diseases of the respiratory organs. The Leipsic Sick Benefit statistics show a very high sickness rate among hatters, viz., 1124 days per annum per 100 members, and a mortality hazard of 1.15 per 100 members.

Preventive Measures.—The chief danger in this industry lies in the employment of mercury and much attention has been paid to provide a suitable substitute for the acid nitrate of mercury. Lussigny suggested the use of a solution which has for its base caustic potash. This preparation has been patented and is said to have been successfully employed in New York, and by some firms in Russia, but apparently failed to give satisfaction in France. Ronzat has urged the substitution of tin for mercury, and this process is now being tried in France, but reliable data concerning the employment of these substitutes are still wanting. Until a harmless substiute for mercury is generally adopted, adequate exhaust ventilation in all processes involving the evolution of dust and fumes should be insisted upon. This, together with the wearing of rubber gloves by those handling mercurial solutions, will at least diminish the dangers. Fortunately, the manufacture of wool-felt hats has displaced to a considerable extent the use of rabbit fur.

In July, 1915, Commissioner Lewis T. Bryant of the Department of Labor of the State of New Jersey published "Sanitary Standards for the Felt Hatting Industry." The report very properly emphasizes the chief health hazards, such as mercurial poisoning, dust, humidity and other injurious environments, which have combined to produce a large number of cases of tuberculosis of the lungs. He states that the industry is regarded so dangerous to health that several of the largest insurance companies refuse to insure fur cutters, makers, starters and sizers at any premium, and workers in other departments are charged an extra heavy premium on ordinary policies.

The general preventive measures and devices recommended in the

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Bulletin have been tried out in different factories and proved of value in reducing to a minimum the industrial hazards. Among the important measures recommended are that all mixing should be done mechanically in an inclosed device, and the fur mixture should be fed into the "Devil" by means of an inclosed automatic device.

Blowing machines should likewise be fed by means of an automatic device, and all dust and fur fiber liberated during the blowing and cleaning operations should be removed by means of the exhaust system, standardized in the Bulletin. Vacuum or wet cleaning only should be employed in the dusty departments.

The temperature in the blowing room and mill room (when due to artificial heat) should not be permitted to exceed 75° and when live steam is employed to increase the humidity, excess temperature should be prevented by means of a cold-water spray or other efficient methods. All steam generated in the operations of starting, first and second sizing, dyeing and blocking should be controlled and eliminated by means of exhaust ventilation. Wood alcohol fumes should not be permitted to pollute the air of the workrooms either during the shellacing or drying operations. Efficient means for their removal should be installed, and care exercised to safeguard against the hazard of explosion. All steam pots and singeing flames should be hooded and supplied with exhaust ventilation. All dust generated in the operation of pouncing and sandpapering machines should be removed by exhaust ventilation. All hand or mechanical ironing shells should be heated by electricity. Wherever gas is employed for ironing purposes the health of the worker must be safeguarded by means of exhaust ventilation. To control the excessive heat generated in the flanging and matricing operations, ample ventilation by means of windows, wall fans or other devices should be provided; all steam plates should be covered on bottom and sides with asbestos, and whenever possible an electrical device for heating the flange-bags should be installed.

Standard safeguarding should be installed on all shafting under power-driven serving machines, and on all belts, gears and wheels; and all set screws should either be replaced or countersunk.

General Preventive Measures for Workers in Mercury.†—1. The imperative necessity of providing local exhaust ventilation, wherever dust and fumes are evolved, as well as a reduction of working hours during the warm weather should be generally recognized.

2. All processes involving the use of mercury should be carried on in separate rooms, with a northern exposure of the windows, preferably at a temperature below 60°, so as to reduce the danger from volatilization of the metal to a minimum. Work should be suspended when the temperature exceeds 78°.

3. Wooden floors and work benches are objectionable as they favor the lodgment of spilled mercury in cracks and crevices. Enameled iron benches

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and smooth asphalt floors, provided with an incline and channels toward receptacles in which the mercury may collect should be chosen. The receptacles should be covered leaving only a narrow opening just sufficient for the metal to enter.

4. Absolute care to prevent spilling of the mercury should be exercised The work benches should be freed from the metal upon cessation of work, the floors should be sprinkled and swept two or three times a day.

5. The workers should be supplied with respirators in all dust-producing processes; overalls and a suitable head covering of a perfectly smooth material should be used and washed weekly. The hair should be worn closely trimmed, preferably no beard; female and youthful employees should be excluded. The use of alcoholic beverages and tobacco should be prohibited.

6. Cleanliness of person and clothing are of the utmost importance. No food should be taken in the workrooms and in no case until after thorough washing of the face and hands with soap and water, using a brush for the finger nails, also washing the mouth and teeth with brush and water, followed by the use of a mouth wash and gargle with a solution of either chlorate or permanganate of potash or phenate of sodium. Heucke recommends the use of akremin soap, containing soluble alkaline sulphides, and believes that the wash water should also contain potassium sulphuratum so as to convert the mercury into insoluble sulphides.

7. The firm should provide not only all the necessities referred to but also the facilities for warm shower and tub baths, suitable lockers for clothing, lunch rooms, and periodical medical examination of the employees, with suspension from work if any are found to present symptoms of mercurial poisoning.

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SECTION VI

PROCESSES INVOLVING EXPOSURE TO POISONING FROM CHEMICALS, INCLUDING BENZOL AND NAPHTHA

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Sulphuric Acid. Hydrochloric Acid. Nitric Acid. The Chlorine Industry. Iodine and Bromine Compounds. Chromium Compounds. Calcium Nitrate. Manganese. The Coal-tar Industry. Benzol. Nitrobenzol. Dinitrobenzol. Aniline. Dimethylaniline. Trinitrophenol. Dinitrophenol. The Petroleum Industry. Dry Cleaning by Benzine, etc. Blanket Stoving. The Paint and Varnish Industry. Manufacture of Oil Cloth and Linoleum. Manufacture of Straw Hats. Manufacture of Matches. Manufacture and Use of Explosives.

The Chemical Industry.—During the last census an average of 24,876 wage earners was employed in this industry in the United States, of whom 23,647 were males, 1063 females and 166 boys and girls under 16 years of age.

Unfortunately we have no reliable American morbidity and mortality statistics. The German statistics cited by Weyl[1] show that among 185,820 employees in this industry in 1905, there were 163,522, or 88 per cent., cases of sickness, with an average duration of 8 days. There were also 11,010 accidents, of which 122, or 1.11 per cent., proved fatal. The majority of the fatal accidents were caused by gases, hot vapors, and the effects of explosions. The statistical data by Leymann[2] show that the laborers and mechanics contributed about 27 per cent. and the chemists 45 per cent. to the morbidity rate from internal diseases, and 37 per cent. and 43 per cent., respectively, to the morbidity from external affections. Diseases of the respiratory passages are especially prevalent among workers in the manufacture of hydrochloric acid, the sulphates, chromates, caustic potash, chloride of lime and trinitrophenol. Diseases of the digestive organs are frequent in all chemical workers, especially among those engaged in the manufacture of hydrochloric acid, soda, sodium sulphates, chromates and aniline and in the regeneration of sulphur. Cases of poisoning are most frequent in the manufacture of aniline. Burns are most frequent in the manufacture of nitric acid, caustic soda, chromates and trinitrophenol. Diseases of the skin are most frequent in the aniline industry and affections of the eye in the regeneration of sulphur.

Curschmann's[3] more recent statistics indicate that the morbidity rate in the large German Chemical works is 54.9 per cent. per annum, with an average duration of 17.8 days. The rate for female workers is 65 per cent. The mortality rate was only 5 per 1000. The morbidity rate was distributed as follows:

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Accidents	16.15
Diseases of the respiratory organs	16.4
Tuberculosis	2.39
Diseases of the digestive system	13.25
Rheumatic affections	8.84
Industrial poisoning	0.4
Industrial skin diseases	0.24

The number of cases of acute industrial poisoning is not so great as is generally assumed. Curschmann has tabulated the cases which occurred in the German Chemical Industry, 1909–1910, and reports 50 cases with 3 deaths and 1 permanent disability in the manufacture of aniline, nitraniline and fuchsin; 29 cases with 1 permanent disability in the manufacture of nitro-compounds; 5 cases in the manufacture of chloranisidin, naphthalein and organic dye stuffs, 4 cases with 1 permanent disability in the manufacture of phosgene; 9 cases from salicylic, oxalic, phthalic acid and pitch; 30 cases in the manufacture of lead compounds; 5 cases in the manufacture of phosphor trichloride; 8 cases from chlorine and bromine; 9 cases with 1 death, and 1 permanent disability from nitrous gases; 12 cases with 1 permanent disability from sulphureted hydrogen; 1 case from ammonia; 5 from chromium compounds; total 167 cases with 14 deaths and 5 cases resulting in permanent disability.

About two-thirds of all the cases of poisoning occurred during the summer, showing that a more rapid evaporation, and possibly the perspiration and exposed skin favored absorption of the toxic agent, especially in the manufacture of dye stuffs and amidobodies. Of the 117 cases of occupational skin diseases in this tabulation, 74 were attributed to the misuse of chloride of lime for cleaning the hands; 23 were produced by benzol, toluol and naphthol compounds, and the remainder by quinine, tar, etc.

Sulphuric Acid.—Sulphur dioxide for the manufacture of sulphuric acid is most generally obtained during the roasting of pyrites and various smelting processes and is converted into sulphuric acid either in the lead chamber or by the contact process. The fumes are very pungent and suffocating and doubtless exert not only irritating but also caustic effects upon the mucous membranes of the upper air passages, and have been known to produce spasm of the glottis. According to Lehmann, sulphur dioxide in the proportion of 0.03–0.04 per 1000 in the air may produce serious effects in persons not accustomed to it, while old hands appear to acquire a certain amount of tolerance. Leymann's statistics show that diseases of the respiratory organs are more frequent in sulphuric acid workers than among other chemical workers. Burns and injuries to the eyes from the acid are not uncommon.

Koelsch[4] reports 12 cases of poisoning by sulphur dioxide and sulphuric acid fumes which occurred in the German chemical industry in 1907. Most of these cases resulted from inhaling large quantities, while entering the lead chambers or acid tanks, or in cleaning out Gay-Lussac towers, or sul-

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phuric acid tank-wagons. In some of these cases it is not always possible to exclude nitrous fumes and arseniureted hydrogen. It is important that sulphuric acid should be free from arsenic. See sulphur dioxide, sulphuric acid, arseniureted hydrogen—list of industrial poisons.

Hydrochloric acid is a valuable by-product of alkali works in the manufacture of sodium sulphate, sodium sulphide, and of soda by the Leblanc and Hargreave's processes. The fumes of the acid are very irritating and, even in small volumes, may produce inflammation of the eyes and of the air passages, bronchial catarrh, coughs, etc. In a more concentrated form they have produced caustic effects on the tips and edges of the tongue, erosions of the teeth, and ulcerations of the nasal wall. When present to the extent of 2 to 5 volumes per 1000 the lungs become distended with blood, and hemorrhages from the lungs and digestive tract are liable to occur. Lehmann considers the safety limit between 0.1 and 0.2 volume per 1000, and relates a case where even 0.5 per 1000 produced a violent reaction.

In the production of sodium sulphate by the Leblanc process there is a possibility of exposure to the fumes of sulphuric and hydrochloric acid in the operations connected with the mufflé furnaces. In the Hargreave's process there may be exposure to sulphur dioxide. There is also a possibility of danger from sulphureted hydrogen during the de-arsenicating process of the acid and in the removal of the tank waste. Burns of the skin from contact with spilled acid are liable to occur and should be promptly neutralized and washed.

According to Leymann the health of employees in this industry is more liable to suffer than in the manufacture of sulphuric or nitric acid. While cases of acute poisoning are rare, diseases of the respiratory and digestive system and defective teeth are comparatively common. Burns from caustic soda occur in 13.87 per cent. of the workers in that department, and serious injuries to the eyes are liable to occur from the spurting of the strong lyes and should be prevented by suitable eye protectors. Diseases of the digestive system are also quite common in those engaged in the manufacture of sodium compounds.

Nitric acid is most commonly produced by the action of sulphuric acid on saltpeter. The decomposition is carried on in cast-iron retorts and condensation takes place in fire-clay Woulff bottles, connected with a coke, or a Lunge-Rohrmann plate tower. The fumes of nitric acid and nitrous fumes, however generated (see list of industrial poisons), are very toxic and the cases often terminate fatally. The fumes do not, except in very concentrated volumes, produce acute and suffocative attacks like those produced by other irritant gases, and as a result the inhalation may go on for several hours and inflict great damage to the blood before the appearance of serious symptoms. The literature contains numerous instances of poisoning caused by leaky retorts, by inhaling the fumes in filling carboys or iron vessels, by spilling of the acid, breakage of carboys and attempts to mop up the spilled acid with

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saw dust. Not infrequently these accidents are the cause of fire and wholesale cases of poisoning have occurred among the firemen as a result of inhalation of nitrous fumes. Rambousek[5] reports an instance where between 50 and 60 carboys stored in an open field caught fire. The 15 firemen experienced no special discomfort during the half hour of exposure, but 3 hours later all had suffocative attacks from which one died and the others recovered after suffering from 9 to 10 days from affections of the respiratory organs.

Schultz[6] reports an instance in which 60 firemen, who were fighting an extensive conflagration in a celluloid factory, suffered from nitrous fumes. The autopsy usually reveals intense inflammation of the mucous membranes of the palate and upper air passages and congestion of the lungs. Czaplewski[7] describes several cases caused by breakage of the carboys by carelessly dragging them along the floors. Lewin[8] discusses the subject of nitrous gas poisoning in the metal industry and proposes excellent methods for their prevention. The branches of industry in which cases of poisoning are liable to occur are enumerated in the list of industrial poisons, see page 734. Apart from the serious effects referred to, the industry naturally involves exposure to burns and predisposes to diseases of the respiratory organs, which constitute, according to Leymann, 12 and 11.8 per cent. respectively of the morbidity.

Preventive Measures.—According to the Massachusetts State Board of Health[9] the corrosive acids are made in such a way that practically no fumes whatever escape, the work being inclosed from beginning to end. In one of the largest chemical factories in that state, where 300 men are employed, it is said that the workmen are exposed very little to poisonous or irritating fumes and dust, or to contact with poisonous or irritating substances. At certain points in the building acid fumes in considerable strength are constantly present, but at these points there is good overhead ventilation, and the workmen are rarely obliged to approach very near. The utmost care is required to safeguard the health of the employees by proper construction of the apparatus and supervision of the various processes, so that there may be no leakage of gases. All such processes should be carried on under official regulations. In the sulphuric acid industry the men who enter or clean out the chamber system, tanks and tank wagons should be provided with smoke helmets. Deposits from tanks should be either scooped out without the addition of water or dilute soda solution, or removed preferably by means of flushing hose without entering the tanks.

In the hydrochloric acid industry it is important that the salt-coke pan and reverberatory furnace is properly constructed. The English Rules provide that: 1. The salt-cake pan must not be charged when overheated. 2. Sulphuric acid shall be added only after all the salt has been charged and the door shut. 3. If hydrochloric acid fumes escape at the door when the Glover acid flows in, the flow must be interrupted. 4. All doors must be closed while work is in progress. 5. Definite times shall be fixed for with-

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drawal of the salt-cake in order to try to ensure that it be not still fuming, but should this be the case, cold sulphate of soda, shall be sprinkled over it.

The Chlorine Industry.—This industry includes the production of chlorine, chiefly from manganese dioxide by the Weldon process, the Deacon process together with its electrolytic generation with simultaneous production of caustic alkali. It also includes the manufacture of chloride of lime, the chlorates and hypochlorites. Chlorine is also used for the production of a number of other chemical compounds which will be alluded to later. In all of these processes there is more or less exposure to the inhalation of chlorine, which is most pronounced in the production of chlorine by the Weldon process, and also in the manufacture of bleaching powder. Fortunately, even the smallest quantities of chlorine cause suffocative sensations which oblige the workmen to leave the rooms and acute cases of poisoning are naturally rare. For symptoms and the industries in which chlorine poisoning may occur see page 727.

Exposure to the irritating fumes of chlorine, even in small volume, cannot fail to predispose to diseases of the respiratory organs. According to Lehmann 0.01 per 1000 in the air is injurious, 0.1 per 1000 produces ulceration of the mucous membrane and 1 or 2 hours' exposure endangers life. Leymann states that the morbidity rates from this class of diseases among men engaged in the manufacture of chlorine and bleaching powder is 17.8 per cent. as compared with 8.8 per cent. in other workers. The so-called chlorine rash or chloracne is believed by Roth and Lehmann not to be due to local external irritation, but to the excretion of chlorine by the skin.

There is also danger from mercurial poisoning, if mercury is used in the electrolytical production of chlorine.

Iodine and Bromine Compounds.—Chlorine is now very generally employed to liberate iodine from the mother liquors of Chili saltpeter and other salt industries. Iodine, apart from its medicinal properties, is used in the manufacture of iodoform and other pharmaceutical and chemical preparations. Ascher maintains that apart from exposure to chlorine gas there is also danger from iodine poisoning, affecting especially the nervous system and not infrequently also resulting in gastric ulcer, the effects being especially marked in photographers who use bromide of iodine in their work. Rambousek[10] cites two cases of acute iodine poisoning in a Swiss chemical factory where organic iodine compounds were made; one proved fatal and was characterized by severe cerebral symptoms, giddiness, double vision and collapse.

Grandhomme[10] has described six cases of methyl iodide poisoning among workers engaged in the manufacture of antipyrine.

The manufacture of bromine compounds involves exposure to chlorine and there is some evidence to indicate that there is danger from bromine poisoning. These compounds are used not only for medicinal purposes, but also in the coal-tar color industry and in photography. Schuler, in 1899 described three fatal cases of methyl bromine poisoning in men engaged in

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preparing this compound by means of wood alcohol and sulphuric acid. In all three patients there were symptoms of nausea, spasms, trembling of the extremities and diminished bodily temperature. These cases were regarded as doubtful, but several cases have since been reported in persons handling ethereal solution of methyl bromide.

Cases of poisoning have also been reported in the manufacture and use of phosphorus chloride, sulphur chloride, zinc chloride, and even rock salt has been known to produce in grinders and packers, ulcers and perforations of the nasal septum.

Among the organic chlorine compounds should be mentioned phosgene, which is employed in the manufacture of different coloring material. Rambusek[10] cites five cases of industrial poisoning and Grempke[11] reports three fatal cases which were caused by the escape of this very toxic gas from leaky rubber tubing during its production.

Methyl chloride, methylene chloride and carbon tetrachloride are also poisonous. In the manufacture of the latter agent the workers are exposed to the possible combined effects of chlorine and carbon bisulphide. In the production of chloroform there is exposure to chlorine and the stupefying effects of chloroform. Chloride of nitrogen is said to be very irritating and is a highly explosive compound. Rambousek mentions that there is some risk from the formation of chloride of nitrogen in the manufacture of gunpowder, if the nitre contains chlorine. Cyanogen chloride is another very poisonous chlorine compound, and besides the danger in its manufacture, Albrecht, cited by Rambousek, has pointed out that it may also be generated in the production of red prussiate of potash, in case the solution is treated with an excess of chlorine. Rambousek reports several instances of industrial poisoning from hydrofluoric acid, some of which occurred during its production, and presented evidence that the vapors exerted a corrosive effect on the mucous membrane of the respiratory passages and of the eyes. Blisters and burns, and even deep ulcers and gangrene of fingers, have resulted from the topical use of the acid in the etching of glass (see also list of industrial poisons).

Preventive Measures.—The same general principles which have been enunciated in the manufacture of corrosive acids apply to this industry and may be summed up in careful supervision of all the apparatus and processes, exhaust ventilation and general cleanliness. It is stated that since the use of "magnetite" instead of carbon for the anode, in the electrolytic production of chlorine, the number of cases of chloracne has diminished. If this be the case it is a strong argument in favor of the belief that the tar cement is a causative factor.

Chromium Compounds.—Sodium and potassium bichromate are extensively used in all of the industries enumerated on page 728.

Cases of poisoning are especially common in the manufacture of the bichromates and may be caused by the dust evolved in the drying, crushing and

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packing of these salts, and also by the vapors from the hot chrome liquors. The air of a crushing room in an establishment, cited by Rambousek,[12] contained from 3.30 to 6.30 mg. of chromate dust per centimeter and of the packing room 1.57 mg. The chrome compounds not only exert caustic effects upon the skin and mucous membranes, but also produce respiratory and digestive disorders and, by absorption, may even inflict serious damage to the kidneys. Eczema and characteristic chrome ulcers are, however, the most common manifestations of industrial poisoning. Leymann's[2] statistics show that of 722 persons engaged in the manufacture of chromates, 252 suffered from chrome ulcers and perforation of the nasal septum; of 257 workers observed by Hermanni[13] 107 had ulcers and 87 perforations. These ulcers are not confined to the nose, but may develop on the tonsils, palate, throat, windpipe, on the arms and hands and other exposed surfaces of the body. The nasal ulcers are doubtless aggravated by picking with fingers. Diseases of the respiratory organs were observed by Leymann in 8.8 per cent. and disorders of the digestive organs in 12.3 per cent. of his cases.

Cases of chrome poisoning have occurred in nearly every industry enumerated on page 728 and next to the manufacture of the salts, the tanning industry and the manufacture of safety matches produce the greatest number of cases. Occasional cases are also reported in the calico-printing and in the dyeing industry, in glass workers and in photographers who employ a bichromate developer, etc. The use of bichromate of lead involves an additional element of danger from lead poisoning.

Preventive Measures.—The machinery used for grinding the raw material, such as chrome ironstone, lime and soda, and also of the finished product, should be impermeable and supplied with efficient exhaust ventilation. The latter is also necessary wherever dust or steam is evolved. Respirators, impermeable gloves, anointing face and hands with vaseline, proper working suits and caps, efficient general ventilation, frequent ablutions, special lunch rooms, baths, and change of clothing upon cessation of work are recommended.

Frequent medical inspections, prompt treatment of skin affections and chrome ulcers, change of work, and a constant educational campaign will do much toward the prevention of disabilities.

Calcium Nitrate.—According to Koelsch,[14] workers in factories where calcium nitrate is produced electrically from the atmosphere are subject to a peculiar form of industrial poisoning, which is atttributed to calcium cyanide in the dust inhaled. The symptoms do not develop until some alcoholic beverage is taken; then the head becomes hot and flushed while the limbs feel cold. Pulsation is felt in the chest and neck, while respiration becomes difficult. Even minute quantities of alcohol may produce these symptoms. The face and neck then become cyanosed while the trunk and arms acquire a bright red-colored rash, unaccompanied by any great rise

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in temperature. The reflexes remain normal, and the senses are unimpaired. In severe cases giddiness and jactation occur. The duration of the attack is variable, according to the amount of alcohol taken. When vomiting or diarrhea occurs, instant improvement follows, and the rash immediately fades. No permanent ill effects have been observed hitherto. No case of acquired immunity has been recorded. A few hours' stay in the dusty factory is sufficient to render a workman liable to attack. Total abstainers are not affected.

Manganese.—Manganese dioxide or brown mineral occurs chiefly as a pyrolusite and in this country is mostly obtained from an ore of which zinc is the principal product. The ore is run through crushers and screens and by means of magnetic belts those containing iron and manganese are picked off, by the belts, and deposited in bins. The crushing, grinding and sifting is a dry process and the workers are exposed to the inhalation and swallowing of this dust. Cases of manganese poisoning have been reported as incidents of this process, and in the manufacture and use of umber colors for mosaic work. Cases of poisoning have also been reported in chemical factories, among men who were engaged in the drying of Weldon mud, the dioxide of manganese having been used for the recovery of chlorine by the Weldon process. Manganese is also used for the production of potassium permanganate, and in the manufacture of glass, as a coloring and decolorizing agent; it is likewise employed in the ceramic industry for coloring and glazing purposes and as a drier in the varnish and oil industry. The literature contains only between 25 and 30 cases of industrial manganese poisoning, but it is highly probable that such instances are much more frequent than is commonly assumed. It is believed that manganese and its compounds, in the form of dust, especially when rich in manganese protoxide may produce the intoxications described on page 119.

Apart from the nervous and locomotor disturbances described by Casamajor and other writers, Neisser[16] refers to headache, dizziness, loss of appetite, constipation, loosening of the teeth, lancinating muscular pains and general debility, as symptoms observed in some of the workers in manganese. The possibility of a combination of manganese and zinc poisoning should be considered.

Preventive Measures.—Improved methods for the arrest of dust at the place of origin by inclosed machinery, its efficient removal by exhaust fans, together with respiratory masks, suitable clothing, facilities for ablutions, mouth washes and gargles before meals, supplemented by shower baths and change of clothing upon cessation of work, should be insisted upon.

COAL-TAR DISTILLATION PRODUCTS OR DERIVATIVES

The manufacture of coal-tar products affords employment to a large number of wage earners, who are exposed to more or less dangerous agents.

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Coal Tar.—Coal tar is obtained by dry distillation of coal and is an important by-product of illuminating gas and coke works. It is chiefly used for the production, by fractional distillation, of so-called coal-tar products, such as benzene, toluene, naphthalene, anthracene, carbolic acid, pyridine, etc., from which again are made saccharine, various drugs, dyes, perfumes, etc. About 50 per cent. of the coal tar is pitch and remains in the stills as a residue. This substance, like tar, is used in the manufacture of varnishes, metal paints, artificial fuel (briquets), etc. Crude tar is also used for preserving wood in the manufacture of roofing paper, in the construction of asphalt pavements, etc. The constitutional effects of inhalation of tar vapors are such as loss of appetite, nausea, diarrhea, headache, vertigo, catarrhal conditions of the upper air passages. In some instances, however, ischuria, strangury, albuminuria and œdema have developed, rarely of a serious nature. Affections of the skin, such as eczema, pimples and psoriasis are quite frequent among workers in tar, and may affect not only the hands and arms, but also other parts of the body. Cancroid ulcers, especially of the scrotum, are liable to develop not only among workers in the tar, lamp black and paraffine industry but also in chimney sweepers and briquet makers.

Benzol.—This important coal-tar derivative is secured by fractional distillation of tar in wrought-iron stills. Crude benzol consists chiefly of benzene and toluene. Pure benzene and pure toluene are secured by additional fractional distillation. Benzol is placed on the market as 90 per cent. and 50 per cent. benzol, and as solvent naphtha. Pure benzene vapors are very poisonous. The limit for animals is between 0.015 and 0.016 per 1000. For symptoms and branches of industry in which benzol is employed and poisoning may occur see page 724.

Dr. Rambousek[16] reports 34 cases of benzol poisoning of which 24 were acute cases and proved fatal. About one-half of the cases occurred in connection with benzene plants, either during the distillation process, or during the cleaning out of the extraction apparatus, or storage and transportation tanks. Twelve cases occurred in the rubber industry, one in the manufacture of antipyrine; one in a painter while applying a coat of asphalt paint to the interior of a large iron tank, in which benzol, instead of turpentine, was used, and three cases occurred in a color factory, from the internal use of alcohol denatured by the addition of 1 to 2 per cent. of benzol.

A case[17] of chronic industrial poisoning by *xylene*, a homologue of benzol, is referred to as having occurred in the rubber industry; also a case of carbon monoxide poisoning and several from sulphureted hydrogen gas in coal-tar distillation.

In the coal-tar distillation, at a temperature of 150–200°C., the middle oil passes over. This contains naphthalene, which upon cooling crystallizes out, and after washing with caustic soda and acid, is redistilled and hot pressed. From the remaining liquor, phenol or carbolic acid is obtained.

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This serves as the basis for various chemical and pharmaceutical preparations, such as salicylic acid, picric acid, etc. The heavy oils which pass over at a temperature between 200 and 300°C. are rich in cresols, naphthols, naphthalene and fluid paraffine, from which lysol and other disinfectants may be obtained. But the bulk of the heavy oils are used for preservation of railway ties, shingles, etc. Raw anthracene oil, or green oil, passes over between 300° and 400°C. and is also used as a wood-preserving paint or stain. The alizarin dyes are made from the anthracene extracted from this oil. Workers in anthracene are subject to severe skin affections which occasionally develop into cancer (Rambousek).[18] Cases of industrial phenol poisoning are said to be rare. Apart from the effects upon the skin, Weyl[19] states that chronic symptoms, such as vomiting, difficulty in swallowing, increased flow of saliva and chronic nephritis, have been observed among surgeons and workers in the manufacture of carbolized dressings. Pyridine is used for denaturing alcohol and the eczematous condition of the skin in persons using denatured alcohol is probably caused by pyridine.

The coal-tar color industry is based upon the employment of the hydrocarbon compounds such as benzene, toluene, xylene, naphthalene, anthracene, phenol and cresoles. Other agents employed are wood alcohol, ethyl alcohol, phosgene and formaldehyde, nitric acid, sulphuric acid, caustic soda, chlorine and bromine. All of the nitro derivatives are obtained by the action of a mixture of nitric and concentrated sulphuric acid on any one of the hydrocarbons of the benzene series.

Nitrobenzol, also known as myrbane oil, or imitation bitter almond oil, is an intermediate product in the manufacture of aniline, and is secured by the action of sulphuric and nitric acid upon benzene, thus forming nitrobenzol. Toluene and xylene are nitrated with the same acids. The dinitro products are obtained by further action of nitro-sulphuric acid on the mononitro-compound at a higher temperature. All nitro-compounds of benzol and its homologues, such as dinitrobenzol, dinitrochlorobenzol, nitrotoluol, nitrophenol, nitronaphthalene, etc., are more or less poisonous. Cases of poisoning from these agents have been reported in the coal-tar industry and establishments in which its intermediate products are manufactured or handled, such as in the manufacture of aniline, explosives, perfumery, soap, pharmaceutical preparations, etc.

Grandhomme[20] has reported 60 cases of industrial poisoning by nitrobenzol, of which 24 or 42 per cent. proved fatal; 4 cases resulted from inhalation and in 55 cases the agent entered the system. Dr. S. S. Adams[21] reports 15 cases with 3 deaths in which the agent was taken into the mouth; 6 cases with 2 deaths were caused by inhalation; 2 cases, 1 fatal, in which nitrobenzol was absorbed by the skin; 2 cases, 1 in which the agent was used with suicidal intent; and 3 cases with 2 deaths in which nitrobenzol was taken to induce abortion. Of the 28 cases, 9 were typical occupational intoxications.

Dinitrobenzol is the combustible ingredient of roburite and other high

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explosives. Oliver[22] reports a case in which a man engaged in cleaning out the air flue, from which the fumes from three mixing pans were discharged, in spite of respirator and rubber clothing, was fatally injured by the fumes. The majority of workers in dinitro-compounds in Great Britain show evidence of chronic intoxication. Of 20 examined in explosive works, 14 presented a bluish-gray color of the lips and face, 10 were profoundly anæmic and 6 suffered from slight tremblings and other nervous affections.[23]

Aniline, also known as aniline oil, is a colorless fluid which upon exposure to light and air gradually becomes dark in color. It is produced by the action of hydrochloric acid and iron filings upon pure nitrobenzol, and like all other compounds of benzol and its homologues, such as toluol, naphthalene, xylol, is a blood and nerve poison. Poisoning may occur in the manufacture of aniline and its derivatives, such as aniline dyes, pharmaceutical preparations, photographic materials, etc., and in dyeing establishments. The poison may be absorbed through the skin by direct contact or from saturated clothing, by inhalation of vapors and impalpable dust, and also by way of the digestive tract. The symptoms may be acute, subacute and chronic, and will be described on page 721.

Dimethylaniline is produced by heating aniline, aniline hydrochloride and wood alcohol. *Diethylaniline* is prepared in the same way except that ethyl alcohol is used. The *nitroso-compounds* are produced by the action of nitrous acid (sodium nitrite and hydrochloric acid) on the acid solution of diethylaniline.

Trinitrophenol or picric acid is produced by the action of nitro-sulphuric acid upon phenol. In a pure state it forms pale yellow crystals and is extensively used as a yellow dye stuff, and also as an ammonia compound in the manufacture of explosives (lyddite, melinite). It enters the body in the form of dust, by inhalation, and also exerts an irritant effect on the skin, mucous membrane, intestinal canal and upon the kidneys. *Dinitrophenol* may cause acute symptoms of industrial poisoning similar to those observed from nitrobenzol.

Leymann, cited by Rambousek,[24] describes cases where the workers were suddenly stricken with symptoms of collapse, vomiting, pains in the chest, difficult breathing, rapid pulse, convulsions and death within a few hours. The autopsy revealed a yellow substance with picric acid reaction, which appeared to be di- or trinitrophenol. In cases of nitrochlorobenzene poisoning Leymann noted the typical gray-blue discoloration of the skin, and the chocolate-brown color of the blood produced by methemoglobin.

Space will not permit reference to other coal-tar color products such as the azo dye stuffs, anthracene colors, alizarin dyes, indigo, fuchsin, methyl violet and sulphur dyes.

Résumé of the Dangers in this Industry.—Much has been done in the last two or three decades to clear up many misconceptions as to the dangers attending the production of aniline and other coal-tar colors. According

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to Grandhomme[1] and Curschmann,[26] the raw product benzol probably caused the greatest number of cases of industrial poisoning. The statistics are not wholly clear since a great many authors fail to differentiate between petroleum and coal-tar benzene. The manufacture of the intermediate group, especially the nitro- and amido-compounds, involves considerable risk. Curschmann mentions particularly nitrobenzol, chloronitrobenzol and dinitrochlorobenzol. Grandhomme[25] has observed no injurious effects from naphthalene and anthracene and considers the danger in the manufacture of fuchsin, of the blue dahlias, greens, resorcin, eosin and the alizarin colors and pharmaceutical compounds comparatively slight. He mentions arsenic and arseniureted hydrogen as possible sources of danger in the manufacture of fuchsin and of the methylene blue, and reports 42 cases of aniline poisoning in one of the chemical works within 3 years, with no fatalities. Professor Roth[27] refers to the fact that in 1905 not less than 38 cases of diseases of the bladder, mostly sarcoma and cancer, occurred among the workers in a German aniline factory in the vicinity of Frankfort. Schwerin claims that tumors of the bladder are quite common in that vicinity, but on the whole it must be conceded that, since the poisonous agents are largely eliminated through the kidneys, the irritant effects upon the mucous membrane of the bladder cannot be questioned, especially since strangury is a frequent symptom in chronic anilismus. It is very likely that some of the skin affections are also caused by the elimination of the poisonous substances through the perspiration.

Cases of aniline poisoning are not infrequent in other industries, especially in dye works. Lymann[28] reports four fatal cases of poisoning in the manufacture of black dyes from mono- and dinitrophenols and nitrochlorobenzene and toluene compounds.

White and Sellers[29] report a fatal case of acute aniline poisoning in a worker who, while preparing aniline hydrochloride (used for dyeing yarn with aniline black), spilled a quantity over his hands, face and upper part of the body, and died within less than 24 hours. Neisser[30] reports a number of cases of aniline poisoning which occurred in different industries, such as wood staining in imitation of mahogany, and describes the case of a Swiss dyer who, in opening an iron drum, spilled a quantity of aniline oil which saturated part of his clothing. He continued work for several hours. The case resulted in insanity and impairment of vision. Cases of anilism have been reported in black dyeing establishments and in cloth pressers working with black dyes.

Senn[31] has reported a typical bronzing of the skin with a dark brown discoloration and inflammation of the cornea occurring among men exposed to the hot vapors from an aniline black dye mixture, composed of aniline, hydrochloric acid and potassium chromate, known as "chinon." Hanke[32] has observed cases of inflammation and opacity of the cornea caused by continued exposure to the vapors of nitronaphthalene or to spurting of the liquid into the eye.

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Preventive Measures.—The coal-tar industry requires careful technical and medical supervision to safeguard the health of the wage earners. The distillation of tar and the purification and cooling of the distillation products must be carried on in properly constructed stills, apparatus and vessels provided with facilities for the removal of injurious gases, and all leaky or defective apparatus should be promptly remedied. In the coal-tar color industry we have to deal not only with the toxic properties of the raw material such as benzene and toluene, but also with the intermediate products such as nitrobenzol, aniline, toluidine. Many of the processes involve exposure to chlorine, acids, especially nitric acid, and sulphuretted hydrogen gas and other chemical agents, which must be guarded against by enclosed processes and mechanical suctional ventilation for the removal of toxic fumes and dust. In addition to these devices, the general ventilation of the establishment should be perfect. All sweeping and cleaning should be done by the vacuum system. General and personal cleanliness are of the utmost importance. The men should wear suitable eye protectors, work in special suits, frequently washed, and eat in special lunch rooms. Frequent ablutions of the hands, especially before meals, and a bath with change of clothing upon cessation of work are essential. The workmen should receive systematic instruction in the prevention of accidents, in first aid, the employment of respirators, pulmotors, oxygen inhalation etc. These measures, with careful periodical physical examination of the employees by a full-time salaried physician, have proved in large establishments of immense value to all concerned. Curschmann, with his large experience in the "de Greppin works," also emphasizes the importance of good wholesome food, abstinence from alcoholic beverages, sanitary homes for the wage earners and a careful selection of the employees engaged in this industry.

The Petroleum Industry.—During the last census year the number of persons in the U. S. engaged in the refining of crude petroleum was 16,640, of whom 159 were women and 43 persons under 16 years of age.

Employees in the petroleum industry are exposed to the gases evolved from the oil, namely, benzine, or petrol naphtha. The men near the wells and pumps and those who enter the tanks or clean out the retorts are especially exposed to concentrated petroleum vapors.

Refining of Crude Petroleum.—Hayhurst[33] states that in receiving the crude product, usually by pipe lines, there was an odor of hydrocarbons (methane ?) and of hydrogen sulphide present in some places, but not hazardous except when leaks occurred, and that cases of sore eyes were not infrequent. The distilling and refining of crude petroleum includes purification with sulphuric acid and its neutralization with caustic soda, de-colorization with Fuller's earth and de-odorization with hydrochloric acid and chloride of lime, and hence involves exposure to these industrial poisons.

The residuum of American oil is especially rich in paraffine, vaseline and other lubricants which are extracted by different processes. In some places

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where the retorts are opened for the removal of the residue there is considerable exposure to sulphur dioxide, and in the recovery of tar there is generally exposure to sulphuric acid fumes. Lead oxide is also added to the oil and the men who shovel it into the open vats and those obliged to repair lead-lined tanks are exposed to the risk of lead poisoning. There is also considerable exposure to dust in the de-sulphurizing process, in which a mixture of copper, lead and iron oxides is used.

Filtering and Pressing.—In this department the residue is separated into heavy oils, greases and paraffine. The work is carried on at a temperature of 50°F. The paraffine is extracted by means of big presses and conveyed by machine work to a chamber where it is heated and filtered. Exposure to ammonia fumes from the cooling plant is noted in the Ohio Survey, and also a vile stench from the boiling down of wool greases to be added to the lubricating oils.

It is very likely, as pointed out by Dr. Wm. H. Sharp,[34] that the toxic effects of petroleum vapors vary with the character of the oil, heavy oils producing mild attacks while the light oils may result in asphyxia. Some of the crude oils cause irritation of the respiratory passages, while others affect the central nervous system.

Petroleum as a fluid has a direct action on the skin, and persons engaged in the filling of barrels, emptying retorts, and handling of paraffine and other residues are liable to develop obstinate inflammatory conditions of the skin—the so-called petroleum and paraffine itch—also pimples, pustules, boils and warty growths. Mitchell[35] was among the first to call attention to these skin affections. Similar effects are produced in furniture polishers, machine oilers and all others who handle either crude petroleum or paraffine, unless protected by impermeable gloves. The literature contains numerous references to cases of poisoning among persons employed in petroleum and naphtha tanks, in cleaning out railway oil tanks, in the use of petroleum motors and in the refining of naphtha, as a result of inhalation of the light oils, benzine and gasoline. The employment of benzine as a motive power, as a cleaning agent, in dry-cleaning establishments, as a solvent of rubber and fats, and as a substitute for turpentine in the manufacture of paints, varnishes, lacquers, etc., is constantly widening the chances for acute or chronic forms of petroleum and benzine poisoning.

Berthenson[36] deplores the scarcity of reliable morbidity statistics in this industry, but reports that of 8465 employees treated at Baku (Russia) there were 1216 cases of diseases of the skin and subcutaneous cellular tissue, 696 burns, 1475 cases of catarrhal affections of the upper air passages, 34 cases of croupous pneumonia, 29 of catarrhal pneumonia, 36 of pleurisy, 5 of pulmonary tuberculosis, and 607 cases of muscular rheumatism. He also cites Petkewitch's observation that quite a number of employees purposely exposed themselves to the intoxicating vapors of petroleum. The German statistics presented by Weyl[37] show that of 1380 employees in petroleum refineries

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9 cases of poisoning and 34 cases of acne were reported in the course of a few years.

Preventive Measures.—The utmost care should be taken to secure efficient removal of concentrated petroleum vapors from tanks and stills by means of compressed air, steam or oxygen. Careful supervision of the work, impermeable gloves, personal cleanliness by means of frequent ablutions of the hands, shower baths, and change of clothing upon cessation of work are indicated. Suspension of work upon appearance of toxic symptoms should be insisted upon. (For further details and preventive measures see page 136, 735.)

Dry Cleaning by Means of Benzine, Chemicals, Etc.—So-called "chemical cleaning establishments" are often carried on in connection with dye works which make a specialty of renovating wearing apparel, etc. The processes and chemical agents employed are quite varied. Apart from the dyes mentioned on page 655, various iron salts such as acetate, nitrate and sulphate, and copper sulphate are used. Alum is employed as a mordant and permanganate of potassium as an oxidizing agent; acetic, hydrochloric, oxalic, and sulphuric acids are likewise employed. Borax, caustic potash, soap and sodium bisulphide are used for washing and bleaching purposes.

The principal agents for the removal of stains are benzene, naphtha, wood alcohol and ammonia; potassium cyanide removes silver stains and oxalic acid removes ink and rust stains.

In the cleaning process soiled gloves and garments are usually placed in a large, revolving, hermetically closed cylinder containing either naphtha or benzine, with or without the addition of soap. After the first cleaning the articles are placed in naphtha alone. After the second rinsing the clothing is placed in a centrifugal machine for the removal of surplus benzine, etc., and then taken to the drying room which is kept at a temperature of between 100°–120°F. On account of the inflammable character of the benzine vapors present extreme caution is necessary, in all processes, to avoid naked flames, matches or other igniting agents. The naphtha fumes in the drying department in spite of mechanical ventilation are very strong, and according to the Massachusetts State Board of Health,[38] "the men employed are pale and some of them markedly sick looking;" some of the men are occasionally overcome by the fumes, or have shown the characteristic excitement and hysterical symptoms of benzene or naphtha intoxication. The presence of vaporized gasoline is frequently noted in the pressing department, where the irons are heated by means of gasoline gas.

Carpet Cleaning.—Some of the establishments also conduct a rug and carpet cleaning department, which is usually equipped with an enclosed dusting machine, exhaust fans and dust flues. The introduction of vacuum cleaners has rendered this operation less dusty and harmful, but the danger from the employment of benzine and benzol for the removal of stains has not diminished.

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Apart from the cases of poisoning already referred to on page 137, 543, Dr. Neisser[39] reports an instance in a carpet cleaning establishment of Berlin where large quantities of benzine had been used. Three of the workmen were found unconscious upon the floor and were with difficulty resuscitated by the inhalation of oxygen.

"Blanket Stoving."—This occupation is of interest chiefly because the blankets are bleached by means of sulphur fumes. It is generally held that the men who are engaged in this work are not especially liable to tuberculosis and other infectious diseases, but those exposed to the inhalation of sulphur fumes and to abrupt changes in temperature not infrequently suffer from chronic bronchitis and emphysema of the lungs.

Preventive Measures.—It is needless to insist that wherever benzine and benzol vapors are developed they should be immediately removed by adequate exhaust ventilation. This is demanded not only in the interest of wage earners, but also as a safeguard against fire. All machines containing benzine or benzol should be furnished with hermetically closed covers, to be removed only for such time as may be necessary to feed or remove the articles to be washed. Substitution for benzine of less toxic agents, such as carbon tetrachloride, has been urged (Rambousek[40]). The same general principles apply to the removal of dust or sulphur fumes in carpet cleaning and blanket bleaching establishments.

The Paint and Varnish Industry.—During the last census 21,896 persons were engaged in the paint and varnish industry of whom 19,496 were males and 2400, or 11 per cent. females. The statistics cover the returns of two classes of establishments, viz., those engaged primarily in the manufacture of pigments or paints and those engaged in the manufacture of varnishes, japans, lacquers or fillers. The principal products of the latter class are mainly solutions of gums, resins, asphaltum, or other ingredients, in such solvents or vehicles as turpentine, linseeed-oil, alcohol, benzine, benzene, etc.

The Massachusetts State Board of Health[41] in discussing the manufacture of paints, colors and varnishes, points out that much of the work is done by men, who have worked from 6 to 20 yeas; the man who makes the lead colors has worked 17 years without sickness. The last case of poisoning occurred 16 years ago, when a number of inexperienced men were poisoned with Paris green. In a color and mordant factory, where aniline colors, logwood, starch, sodium dichromate, etc., are used, about one in five of the employees was noticably pale and sallow, and inflamed eyes, caused by the sodium dichromate, were not uncommon. In the manufacture of "whiting" about half of the 58 men employed in three establishments visited "looked to be in poor condition." In making *shingle stains* pigments, like chromate of lead, Prussian blue, zinc and iron oxide, are first mixed or ground with linseed-oil or with creosote or some of the "heavy or light oils." In two of the establishments the men appeared to be careless in the matter of handling the pigments.

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Manufacture of Linseed-oil, Dryers, Varnishes, Japans, Etc.—Linseed-oil is extracted from linseed previously heated and crushed and submitted to 3500 lb. pressure in hydraulic presses. The temperature in the press rooms often reaches above 125°F. Refined linseed-oil is obtained by treating the raw and filtered oil in lead-lined tanks with a 10 per cent. solution of strong sulphuric acid. Dryers and fillers are usually made by boiling linseed-oil with some oxidizing substance, such as oxide of lead, manganese, etc. During the boiling of linseed-oil, acrolein vapors and considerable heat are evolved, which should be carried off by means of hoods and exhaust ventilation.

Varnishes are solutions of various gums and oleoresins dissolved in alcohol and turpentine. Unfortunately, quite commonly, benzene, benzine and wood alcohol are substituted as solvents. Among the many varnishes may be mentioned asphalt, Burmese, copal, dammar, mastic, spar, and wax varnishes. Shellac dissolved in alcohol or wood alcohol is most frequently used for wood work. Thompson[42] states that orpiment is occasionally employed to make poor grades of shellac yellow and opaque. Copal and asphaltum varnishes are most commonly employed as a lacquer for metal work. Zapone lacquer, which is a solution of celluloid in amyl acetate and acetone, is employed in various industries, chiefly as a lacquering agent in metallic ware and jewlery factories. Unfortunately, we have no morbidity and mortality statistics of these industries.

The Ohio Survery of 1914 by Hayhurst covered 40 establishments with 757 men engaged in these processes. The investigation disclosed 22 positive and 4 tentative cases of lead poisoning, and 5 positive cases of benzine poisoning. Among the common personal complaints were nausea, loss of appetite, dizziness, restless sleep, trembling, palpitation, frequent urination, headache, constipation, eczema and inflamed eyes. All of these symptoms can be reasonably attributed to the effects of injurious dusts, fumes and vapors.

Preventive Measures.—Acrolein, amyl acetate, benzene, benzine, lead compounds, manganese, pitch, turpentine and wood alcohol are industrial poisons, and the workers should be protected by adequate ventilation during not only the manufacture but also the use of the agents containing them. It should also be remembered that all quickly drying spirit or inflammable paints and varnishes are not only a source of benzine and wood alcohol poisoning, but may be the cause of serious burns from ignition of the vapors by a naked flame. (See also painting.)

Manufacture of Oil-cloth and Linoleum.—This industry is inimical to health on account of a number of injurious factors connected with the different processes of its manufacture. Linoleum is made by spreading a mixture of oxidized linseed-oil, resin and ground cork over a rough ground texture of cloth, which is varnished on its lower surface. It is made either in one color or in patterns. The patterns according to Doehring[43] are pro-

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duced in three ways: (1) By the application upon the ground texture of a mixture of covering masses of different colors and grain; (2) by the juxtaposition and fastening of differently colored and arranged patterns upon the ground texture; (3) by printing the plain linoleum with oil colors. The resulting products are known as grain, inlaid, and printed linoleum. The colors often contain arsenical, lead and mercurial pigments. It may be said that the covering mass of linoleum consists chiefly of an intimate mixture of oxidized linseed-oil (linoxyn) and finely ground cork meal. The latter is obtained from the waste material of the cork industry, cut up and ground into a very fine powder by suitable machines and rollers. This gives rise to large volumes of light floating dust, and although morbidity statistics are not available, there is every reason to believe that it is liable to produce diseases of the respiratory passages. Indeed the cork industry in general, such as making cork soles and corks for bottles has long since been regarded as a very dusty trade. Apart from the danger of dust inhalation cork dust is highly inflammable and explosions have occurred not only during the grinding operations, but also during the subsequent mixing processes.

For the purpose of making linoxyn the linseed-oil to which certain oxidizing substances such as litharge, sugar of lead, oxide of zinc, superoxide of manganese, etc., have been added is usually boiled at a temperature between 392° and 464°F. This may be done in open or hooded vessels; in either case frequent stirring is necessary to secure a thorough admixture of the oxidizing agents, and to prevent excessive heating of the oil on the walls of the utensil. This involves not only exposure to excessive heat and frequent burns but also to the inhalation of acrolein and other industrial poisons. As a matter of fact inflammatory conditions of the mucous membrane of the eyes, nose and upper air passages are not uncommon. When the oxidized linseed-oil is ready for use, it is mixed in a steam-heated vessel with 38 parts of resin, 13 parts of Cowrie or New Zealand gum, and some suitable coloring material. This mass is next thoroughly mixed with an equal weight of cork meal, in a suitable mixing machine. Benzine and turpentine are sometimes used to render the mass more plastic and less brittle. As substitutes for cork, wood flour, pulverized peat or other vegetable fiber have been used and in place of linseed-oil, the distillation residue of palm and cotton-seed oil are employed. The addition of resin to hot linseed-oil develops volatile substances. The ether oil which forms is said to produce headache, nausea and sometimes complete stupor. Diseases of the upper air passages, chronic bronchial catarrh and even hemoptysis may be produced according to Eulenberg by the inhalation of these volatile vapors. Workers in resin are also liable to eczema and other affections of the skin.

The next process in the manufacture of linoleum consists in the application of the covering mass (heated to a temperature between 280° and 300°F.) to the ground texture, by means of plate and roller presses, which are hollow

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and heated by steam under pressure. The other processes are the cooling of the mass on cold rollers and the drying of the linoleum in special rooms at a temperature between 77° and 86°. The pattern printing is usually done by means of forms of wood or metal forms upon printing presses or machines in oil colors which not infrequently contain toxic pigments. The linoleum receiving the patterns is again placed in the drying rooms and when completely dried, it is cut, the upper surface is washed and occasionally receives a coat of zapone lacquer.

Preventive Measures.—Adequate provisions should be made in the way of hoods and exhaust ventilation for the removal of dust and fumes; the latter on account of their offensive character should be conducted to the furnace fires and burnt up, or into the smoke stacks and discharged at great heights. Mechanical stirrers should replace the hand ladles. Since cork dust explosions are often caused by particles of iron-producing sparks in passing through the mills, Doehring[43] recommends passing the cork through a magnetic separator before being placed on the comminuting machine.

Manufacture of Straw Hats.—The employees in this industry are exposed not only to the inhalation of dust and unfavorable working conditions, but also to a number of toxic agents in the bleaching, varnishing, sizing, staining and blocking processes. The stock is most commonly bleached by exposure to sulphur fumes in special chambers, and unless all fumes are driven off before the doors are opened the persons who remove the stock are subject to inhalation of these fumes.

Bleaching is also accomplished by immersion of the stock for several hours in a solution of bisulphate of sodium, zinc dust and hydrochloric acid, or by the employment of a bath of oxalic acid, tartaric acid, acetic acid, sodium peroxide and hydrogen peroxide. After removal of the braid from the bath, the stock is placed in a drying room. The varnishing process with shellac dissolved in wood alcohol, and staining process with aniline in wood alcohol, involves exposure to the toxic fumes of this agent. The persons employed in sizing the hats with glue are exposed to excessive heat, since a temperature of about 100°F. appears necessary "in order that the glue may run off freely." The girls who trim the hats often occupy a stooped position. The general working conditions of all the employees should be improved by adequate air-space, exhaust ventilation and proper lighting. (See also felt hat industry.)

The Manufacture of Matches.—The dangers from chronic phosphorus poisoning in this industry, when white phosphorus is used, has been pointed out on page 145. It is simply a question of time when this danger will be wholly eliminated by the general substitution of red phosphorus, and the manufacture of so-called safety matches.

The ordinary process involves fixing the matches in frames and dipping the ends, first into paraffine or sulphur and afterward in the phosphorus paste, consisting of a solution of gum or dextrine and yellow phosphorus,

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containing also some oxidizing agent such as red lead, nitrate of lead, manganese dioxide or niter. The matches are then dried and packed.

The preparation of the paste, dipping, drying and packing involves the greatest occupational risks and, although all these processes can be carried on automatically by American machinery, it did not prevent to any considerable extent the occurrence of poisoning; hence the act imposing a tax of 2 cts. per hundred upon matches made with white phosphorus which went into effect July 1, 1913, and which is practically a prohibitive act.

The paste for safety matches consists of potassium chlorate, sulphur, or antimony sulphide, potassium bichromate and glass powder, mixed in a solution of gum or dextrine. The matches are saturated with paraffine or phosphate of ammonia. The friction surface for these matches is made up of a mixture of dextrine, antimony sulphide and red phosphorus, the latter, on striking, acts as the igniting agent.

Some authorities maintain that the use of red phosphorus is not free from danger, but in France no cases of poisoning have occurred since the use of white phosphorus was forbidden in 1898.

The manufacture of Swedish safety matches, when chrome salts are used in the paste, has given rise to chrome ulcers and severe eczema. Wodtke[44] reports 18 perforations of the septum nasi among 84 workers. For preventive measures see Phosphorus, page 153; Chromium compounds, page 541.

THE MANUFACTURE AND USE OF EXPLOSIVES

Explosive compounds are used not only for the purpose of propelling projectiles from firearms, but also for the explosion of shells, torpedoes, mines and blasting operations of all kinds. The principal propelling agent employed up to 1845 was gunpowder, but since that time new compounds have sprung into existence, many of which are known as high explosives and fulminates.

According to "Cundill's Dictionary of Explosives," cited by Prof. Munroe[45] there were upward of 1000 explosives in 1895, which have materially increased in number since.

Gunpowder.—The ingredients in gunpowder are 75 to 76 parts of saltpeter, 14 to 15 of charcoal and 10 of sulphur. Slightly different proportions are used in various countries. Gunpowder explodes at a temperature of about 700°F. The force of the explosion resulting from the production of gases expanded by the intense heat developed by the chemical action of the combustible ingredients with the niter, varies with the conditions under which it is fired, the density of the gunpowder and the shape and size of the individual grains.

The force of an explosion from gunpowder may become intelligible when we realize that according to some authorities there are a million foot-tons of energy per second for each ton of powder. Black blasting powder in this country contains about 73 parts of sodium nitrate, 16 parts of charcoal and

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11 parts of sulphur. According to Munroe no marked changes have been made in the manufacture of or composition of gunpowder or blasting powder, but the latter has new competitors in the composition made by mixing finely divided metals, such as aluminum or alloys, ferrosilicon, with potassium or sodium nitrate or other oxidizing agents.

Dr. E. R. Hayhurst[46] investigated seven establishments for the manufacture of explosives in Ohio and the following is a brief summary of his findings. The actual number of persons employed was 303 wage earners of whom 51 were females. The general appearance of workers was fair to good in four places, but a considerable number of unhealthy men were found in some of the processes in two places.

In the grinding rooms some of the men complained that the nitrate caused headaches and dizzy feelings at times, hence the machines should be properly enclosed. The pulverizing of charcoal was exceedingly dusty and complaints of bronchitis were made. The grinding of cakes of finished powder is usually done in isolated places, the workmen being provided with small sheds to avoid danger from explosion while the grinding is going on.

In the incorporating mills the work is extremely dirty and care must be taken to see that the charge does not work out from under the grinding wheels, otherwise the wheel may cause contact with the base and form a spark. Since explosions are especially liable to occur in this process, water is thrown on constantly to keep the charge cool. The work in the press rooms where the pulverized charge is pressed into flat cakes by hand or machine presses is attended with the production of considerable amount of dust. The same is true of the operations of the corning mills, which reduce the powder to the proper-sized grains. The glazing of the grains of powder with lead sulphide and graphite appears to be free from risk of lead poisoning, although some of the workmen had bad teeth. The separating, weighing and packing operations appeared to be free from hazards.

Fulminating powder is used for charging percussion caps and parlor rifle shells, detonators and electric detonators. It is composed of about 35 parts of fulminate of mercury, 16 of chlorate of potassa, 45 of glass dust, 2 of gum arabic and 2 of gum tragacanth. These are mixed by using the fulminate in a moist state. It explodes readily by percussion and by friction or by a heat of 367°F. The manufacture of fulminate of mercury which is made by dissolving mercury in nitric acid and adding alcohol, is attended with the evolution of injurious fumes containing according to Rambousek[47] ethyl acetate, acetic acid, ethyl nitrate, nitrous acid, and volatile hydrocyanic acid compounds.

Apart from the inflammability of these vapors which have been the cause of fires and accidents, "their inhalation is liable to cause headache, vertigo, loss of consciousness, numbness of the extremities and a sense of constriction of the chest which may be accompanied by cyanosis" (Oliver).[48]

In the manufacture of percussion caps, there is also danger of mercurial

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poisoning; about 40 per cent. of the females employed in a Nuremburg factory are reported to have suffered from mercurial poisoning, due according to Heinzerling to the inhalation of mercury fumes developed by tiny explosions in the pressing and filling. Neisser[49] also reports cases which occurred in a factory at Marseilles in 1907.

Preventive Measures.—It is needless to insist that in the manufacture of fulminate of mercury, the dangerous gases like nitrous fumes, cyanogen, and acetic acid compounds are promptly removed by efficient exhaust ventilation. The persons employed should be skilled and realize the danger, both as to explosion and as to poisoning.

Gun Cotton or Pyroxyline.—Ever since its discovery by Schönbein in 1846 attempts have been made to use it as a substitute for gunpowder, because its projectile force, when used in moderate charges is equal to about twice its weight of the best gunpowder. Its explosive force is also very much more intense than that of gunpowder and in this respect its nature is similar to the fulminates. Gun cotton explodes at a temperature of about 277°F. and evolves little or no smoke as the principal residue of its combustion is water and nitrous acid. It is made by immersion of cotton wool in strong nitric and sulphuric acids in so-called nitrating centrifugal machines. The danger from explosion was very great, when drying stoves were employed, but this has been overcome by using alcohol in dehydration. Gun cotton is used in the manufacture of smokeless powder and also as a charge for torpedoes and submarine mines.

Collodion cotton is prepared in a similar way as gun cotton, except that the acids are weaker, and the product is a partially nitrated cellulose. When dissolved in alcohol-ether it is known as collodion, which is extensively used in surgery, photography and in the manufacture of celluloid, artificial silk, gas mantles, moving-picture films and other pyroxylin plastics.

In the manufacture of gun cotton and collodion cotton the chief danger to the workmen are the fumes of nitric acid and nitrous fumes, but with good air-tight machines these risks, and subsequent erosions of the incisor teeth have been greatly diminished. There is also danger from explosions in the drying process as evidenced by the accidents reported by Marshall.[50]

In the manufacture of collodion, celluloid and artificial silk the workmen may also be exposed to ether, alcohol, acetone, acetic ether and camphor, but Rambousek found no cases of such poisoning in the literature of the subject.

Preventive Measures.—In the preparation of gun cotton and collodion, it is important that the acid fumes from the nitrating and centrifugalizing machines are carried off by earthenware ducts and fans, and that the bulk of the acid is removed by centrifugal action. Cleanliness and efficient exhaust ventilation should be insisted upon in all the processes.

Smokeless Powder.—This explosive is derived from gun cotton or other forms of nitrocellulose and is gelatinized by the addition of alcohol-ether, or

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acetone, camphor, resin, etc. The resulting paste or dough is then rolled, washed, dried and pressed into rods. According to Marshall, "the press most commonly used is an adaptation of the machine employed for making macaroni. The resulting product is in the form of cords, strips or tubes, which can be cut into flakes of any desired thickness." It is of interest to note that the first successful smokeless powder was made in 1865 by Captain Schultze of the Prussian Artillery from nitrated wood, instead of nitro-cotton.

Among the numerous kinds of smokeless powder should be mentioned the following: Nobel's nitroleum (artillery powder) which consists of one-half of nitroglycerine and one-half of collodion cotton. "Poudre B" named after General Boulanger and used in the French Army. The "Russian," "Spanish" and "American" powder. In this country according to Schüpphaus cited by Marshall[51] a pyro-collodion powder has been adopted for all kinds of guns. The composition varies but averages 80 per cent. of gun cotton, between 10 to 19.5 per cent. of soluble nitrocellulose, 0.5 to 1 per cent. of urea; some powders also contain 9 per cent. of nitroglycerine. Other varieties of smokeless powder are *Ballistite*, a mixture of 40 to 50 per cent. of collodion cotton, and 50 to 60 per cent. of nitroglycerine, invented by Nobel in 1887.

In order to make the powder more stable or to improve its physical properties it is mixed with such substances as diphenylamine, aniline and calcium carbonate.

Filite is the Italian name for ballistite because it is drawn out into cords instead of flakes. It consists of equal parts of nitroglycerine and collodion cotton with 0.5 to 1 per cent. of aniline. *Tonite* is a mixture of gun cotton and nitrate of barium in almost equal proportions. *Solenite*, also used as a military powder in Italy, is composed of 33 per cent. of nitroglycerine, 1 to 3 per cent. of light colored mineral jelly and the rest of nitrocellulose. The mixture is gelatinized with acetone and the dough is pressed into a tubular form and cut into short lengths. The grains are translucent and resemble light brown glass beads.

The Germans employ a variety of smokeless powder. The "Würfelpulver" is similar in composition to the Italian Filite, but is made up in the form of cubes instead of tubes. *Cordite* is essentially a British invention and is now considered one of the most successful smokeless powders. The composition of the modified form is gun cotton 65 per cent.; nitroglycerine 30 per cent. and vaseline 5 per cent. Marshall credits Col. Sir F. L. Nathan of the British Army with marked improvements in the manufacture of cordite as regards safety of the workmen, stability of the explosive and reduced cost of the product.

Professor Munroe reports that a notable change in practice in the manufacture of smokeless powder in the United States has been the abandonment of the nitroglycerine—nitrocellulose powder by the Army, and the adoption of a straight nitrocellulose powder of definite nitrogen contents as used in

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the Navy. The tendency in military powder is to approach more closely the principle set forth by Munroe many years ago as governing the ideal smokeless powder, viz., that "it should be composed of a single chemical substance in a state of chemical purity."

The smokeless shot gunpowders are manufactured in great variety, in different countries and are broadly divided into the "condensed" and "bulk types." In the former the gun cotton is completely gelatinized and the process of manufacture is similar to that of rifle powder, except that the paste is formed into small grains or thin flakes.

In the production of bulk powder other ingredients such as paraffine wax, starch, lamp black, wood meal, various gums, potassium ferricyanide, nitrates of barium and potassium, bichromates of potassium and ammonia and the mono and dinitro derivatives of benzene and toluene are used in some of the powders.

For a very complete account of the production of smokeless shot gunpowder by Professor Munroe. (See U. S. Census Bulletin No. 92, 1908, page 84, and Marshall's book on Explosives.)

Nitroglycerine was discovered by Sobrero in 1847 and is made by the action of a mixture of nitric and sulphuric acids on anhydrous glycerine.

Professor Munroe[52] states that "by the use of artificial refrigeration the yield of nitroglycerine from a given mass of acid is increased, the speed of nitration is likewise increased, the danger attending nitration is decreased and the use of a second separator is rendered unnecessary." The first attempts to use nitroglycerine in a liquid form for blasting purposes proved so disastrous that a number of countries prohibited its use. Alfred Nobel in 1866 discovered that when mixed with "Kieselguhr" (an infusorial earth) previously heated to redness, it could be rendered less sensitive and more safely handled and transported. This product is now known as *dynamite.* Nobel further discovered in 1875 that by the substitution of gun cotton for Kieselguhr in the proportion of 7 or 8 per cent. of the gun cotton to 92 or 93 per cent. of nitroglycerine, the latter was converted into a gelatinous solid, which he called "blasting gelatine." This blasting gelatine forms the basis of numerous valuable high explosives. Other explosives referred to by A. Cooper Key[53] are *Gelatine dynamite,* which is composed of nitroglycerine thickened by the addition of gun cotton and combined with wood meal, charcoal or certain other non-explosive ingredients. "*Gelignite* a very widely used high explosive is practically gelatine dynamite with the addition of saltpeter. *Carbonite* is composed of about 27 per cent. of nitroglycerine absorbed in a dope of woodmeal and nitrate of potassium or barium." In America, according to Marshall, the formulæ vary considerably, and ammonia dynamites are made in which a large proportion of the nitroglycerine is replaced by ammonium nitrate.

Nitroglycerine can be readily detonated by friction or fulminate percussion caps. It is known to be a strong poison which may be inhaled and can

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also be absorbed by the skin and from the alimentary canal. (For effects see list of Industrial Poisons, page 733.)

Key[53] states that "no one can enter a cordite drying room without experiencing a peculiar sensation at the heart and at the back of the head, which in the absence of fresh air soon develops into a headache which reduces to insignificance all the other ills of life." So far as he could gather no permanent harm resulted from this cause, and on most people the fumes appear to lose their effect after a few days, in others it takes weeks before they become inured and in still others a return to the work after a few days absence causes the original symptoms. Rambousek[54] cites Kobert's case, where the rubbing of a single drop of nitroglycerine into the skin caused symptoms lasting 10 hours.

While Oliver[55] states that women who mix Kieselguhr and nitroglycerine do not suffer, Rambousek[54] reports that workmen engaged in washing out nitroglycerine from Kieselguhr earth, having, in doing so, their bare arms immersed in the liquid, suffered. Other injurious effects especially noted in the mixing and sifting of dynamite are obstinate ulcers under the nails and finger tips and eruptions on interdigital spaces and the plantar aspect of the feet. There is also considerable danger from nitrous fumes unless the nitration is carried on in a closed apparatus. Accidents from this cause are still possible in the acid separating house, wash house and in the denitration room of waste acids. Rambousek reports a fatal case in a workman who tried to wash away with water the acid which had overflowed and inhaled so much nitrous fumes that he died 16 hours later. Among other fatal instances he mentions the case of an employee in a dynamite factory who perished in cleaning out a storage tank for waste acid, in spite of previous swilling and ventilation.

Dr. C. E. Laws[56] gives a very vivid description of the "nitroglycerine head" from which the men employed in dynamite and gelatine factories, miners and excavators so frequently and severely suffer. He states that a man working in powder may cause another person a great deal of misery by simply shaking hands or permitting articles which he has been using to be handled by the other. A worker frequently carries some of the drug in his clothing to his home and may make his whole family sick. Sleeping in the same bed or wearing contaminated clothing will produce effects. Apart from the symptoms described in the list of industrial poisons page 733. Laws states that powder men become easily intoxicated with a tendency to develop acute maniacal attacks and that all who work in it are sooner or later troubled with dyspnœa and tachycardia on exertion; he also states that the drug has a marked aphrodisiac effect and that most glycerine men have large families.

Dr. Evans[57] reports a case of nitroglycerine poisoning in a farmer. The symptoms developed after 1 week of work of blasting stumps with a giant powder of the nitroglycerine variety. Dr. Evans found that the man wore

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cotton gloves which aided by perspiration had become saturated with the powder and evidently caused the symptoms by absorption.

Dr. Geo. E. Ebright[58] in studying the effects of nitroglycerine in those engaged in its manufacture with the Dupont Powder Co. at Hercules, California, found only occasionally persons immune to the effects of nitroglycerine, and that there is a special susceptibility in warm weather. In order to maintain their immunity workers are in the habit of placing some of the products under the hatband, during periods of absence from the factory. The general health appeared to be in no way impaired, but this he attributes largely to the picked character of the men. He also refers to the cordite habit which developed in the British Army during the Boer War. The narcotic effects were accidently discovered. The habitue is able to take the contents of a cartridge, his face flushes, his head throbs and seems to swell and then in about 15 minutes comes a long sleep. On awakening he suffers from intense headache and thirst. When mixed with beer or hot tea it causes a wild delirious intoxication and this is followed by sleep.

Roburite, Ammonite, Securite, Etc.—Reference has been made on page 544 to dinitrobenzol, which is the combustible ingredient not only of the explosives named, but also of other high explosives, chiefly in combination with ammonium nitrate. There are a large number of explosives which contain more or less of dinitro-compounds mixed with a chlorinated hydrocarbon. While dinitrobenzol was formerly most commonly employed, trinitrotoluene is now most extensively used, alone or combined with nitrate of ammonium, potassium or barium as oxidizing agents.

According to Rambousek *ammonite* is a mixture of nitro-naphthalene and ammonium nitrate; *securite* is the same as roburite with the addition also of ammonium oxalate.

A number of cases of poisoning in the manufacture of roburite are cited by Rambousek[59] and also the fact that in the Witten factory almost all the workers between 1890–7 suffered from pallor, blue lips and yellowish conjunctivæ. The morbidity has, however, been materially decreased by efficient preventive measures. Cases of poisoning have also been reported in the nitrotoluene department of explosive plants due to nitrous fumes and mono-nitrotoluene.

The process of grinding, melting, cooling, mixing and fitting in air-tight cartridges is generally well safeguarded; the chief danger is from dinitrobenzol and toluene. (For symptoms and effects, see list of Industrial Poisons.)

Explosions are liable to occur in Benzol plants as shown by the accident reported in Birmingham, Alabama, October 16, 1915.

Picric acid formerly used in the manufacture of *melinite* and *lyddite* is being replaced to a considerable extent with trinitrotoluene. Cases of poisoning from picric acid are rare, although skin affections, vesicular eruptions and yellow pigmentation of the epidermis and of the conjunctiva and digestive derangements have been attributed to this agent.

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Safety Explosives.—There is no such a thing as a safety explosive. The term should be limited, first to those explosives which are comparatively insensitive to blows and friction, such as ammonium nitrate explosives containing no nitroglycerine and hence are safe in handling, and second to those explosives that have been officially tested and found to be comparatively safe for explosion in the presence of fire damp and coal dust. In numerous countries so-called permissible explosives are listed and explosives which do not come up to the required standard cannot be used in coal mines. (For classification and lists of permissible explosives see Marshall's book, 1915, page 472, and Bulletin No. 17, U. S. Bureau of Mines, 1911, page 26.)

In 1875 Great Britain passed a law for the prevention of accidents in the explosive industry, which provided for the appointment of inspectors with power to inspect all magazines and factories and see that the operations are carried on in a reasonably safe manner. As a result the fatalities from explosions have been reduced, according to Marshall, from an average of 43 killed per annum in 1870 to seven in 1913, in spite of a great increase in the number of employees.

The Choice of a High Explosive.—Marshall[60] in discussing this subject states that for blasting *very hard rock*, such as gold quartz, blasting gelatine gives the best results, provided it be fired with a very powerful detonator or primer of non-gelatinized nitroglycerine explosive, such as American straight dynamite or lignin.

For *soft-rock* blasting, gelignite is most commonly used in Great Britain; ammonium nitrate and chlorate explosives are also employed. For *coal-mining* operations Robbinite is most popular. For *agricultural purposes*, such as breaking up hard subsoil, removal of tree stumps, digging ditches, etc., if the charge can be tamped well, blasting powder will answer. If not, a detonating explosive should be used. For *blasting rock under water* a brisant explosive is required and the cartridge should be thoroughly waterproofed, unless the explosive contains a considerable percentage of gelatinized nitroglycerine.

For *military blasting operations*, gun cotton compressed in powerful hydraulic presses, also picric acid powder, trinitrotoluene, and other nitro-derivative explosive compounds are most commonly employed. Smokeless powder and the ordinary commercial explosives are also used by military engineers.

For *submarine mines and torpedo war heads* the requirements are much the same as for military blasting explosives. Nobel is said to have used mines of nitroglycerine to protect the mouth of the Neva during the Crimean War. Picric acid has been used, but this has been to a great extent replaced by trinitrotoluene. During the American Civil War torpedoes containing about 60 lb. of gunpowder were employed in the defense of river sand harbors. On June 4, 1864, according to the surgical history of the war, two large torpedoes

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were accidentally exploded near New Berne, killing 36 soldiers and 8 negroes and seriously wounding 29 men.

Fougasses are small subterraneous mines constructed in front of the weak parts of a fortification. During the American Civil War they were also placed along the roads leading to important points. They are as a rule simply large shells arranged with levers connected with a percussion cap and sunk below the surface of the ground in the supposed path of the assailing party. A pressure of the foot upon the concealed lever is sufficient to explode the shell. In modern times they are exploded by means of electrical current.

For shells, which are hollow iron or steel projectiles adapted for enclosing a quantity of gunpowder or other explosive compounds and designed to be torn asunder upon the explosion of this material, the ordinary black powder was used almost exclusively for a long time. After the discovery of gun cotton, picric acid and other high explosives they were given a trial, and at the present time trinitrotoluene compounds are most commonly used.

A shrapnel is a shell filled with musket balls consolidated into a mass by a composition of sulphur, or the balls are imbedded in melted trinitrotoluene. The shells are exploded by a charge of powder fitted into their interior which may be ignited either by a time or percussion fuse.

Armor piercing shells are expected to pass through the armor and explode when they are on the other side. Many of the mechanical difficulties for the attainment of this object have been overcome. Black powder is still extensively used to fill these shells, as the nitro-aromatic explosives generally detonate on the face of the armor plate. Ammonium nitrate explosives like ammonal also satisfy the requirements, except that they are very deliquescent, which renders their storage for long periods difficult.

Hand grenades are small iron shells from 2 to 3 in. in diameter filled with powder and discharged by percussion. They were first introduced in the seventeenth century, lost for a time their importance but were used not only in our Civil War, but appear also to be extensively employed in the present European War in sieges and assaults at close quarters. Their effects are similar to ordinary shells. At present the explosives most used are picric acid, trinitrotoluene, compressed gun cotton, tonite and smokeless powder. The bombs thrown from flying machines during the present European War are doubtless of a similar nature.

Liquid and Gaseous Projectiles.—During the present war some projectiles of a liquid and gaseous nature appear to have been revived and possibly new ones invented. Among those may be the "carcass," which is a spherical shell having three additional holes of the same dimensions as the fuse hole, and filled with a composition which burns with intense power from 8 to 10 minutes and the flames issuing from the holes set fire to everything combustible within reach. It was chiefly used for incendiary purposes and to light up positions at night. The so-called "Greek Fire," composed of a mixture of coal tar, naphtha, turpentine or coal-oil mixed with phosphorus,

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according to the surgical historian of the Civil War was employed at the siege of Charleston in 1863, but its obvious barbarity led to its speedy discontinuance. The employment of the suffocating gases like chlorine and bromine has been reported in the present war. Among the liquid projectiles may be mentioned the water shell invented by Prof. Abel of Woolwich. The missile consists of ordinary shells filled with water and fixed with compressed gun cotton and a detonator of fulminate of mercury.

Manufacture of Small Arm Ammunition.—We have no detailed statistics relative to the risks attending this occupation, except Key's table,[61] which indicates that of the 175 accidents which occurred in the decade from 1890–99 in Great Britain in the explosive industry, 39 occurred in the manufacture of ammunition, with 5 deaths and 43 injured. Apart from this danger, the workmen are also exposed to industrial poisons and the hazard incident to brass founding, metal working, machine shopping, soldering, wood working, painting and varnishing. (See p. 570). The following is a summary of conditions observed by Hayhurst[62] in his Ohio Survey:

The waterproofing of paper shells by means of hot paraffine was a hot and steamy process. The manufacture of felt and wads for these cartridges was also hot and steamy and attended with a peculiar foul odor due to the dirty cow hair. The work in the lead room and loading and packing of the shells was practically all mechanical and appeared to be free from occupational hazards. The manufacture of copper and brass shells, especially the annealing and washing process, was characterized as hot, wet and humid. The loading and packing of rifle cartridges was a mechanical process, girls operating the machines, and did not touch the lead. In the manufacture of fulminate priming caps the man who did the work was thoroughly skilled and realized the danger both as to explosion and as to poisoning from fulminate of mercury. In the nitroglycerine department the work was also done by skilled men, but some of the helpers, it was said, occasionally complained of headache, flushing of the face and a feeling of distention in the stomach.

Fuses.—A great many varieties of fuses for the ignition of the detonator or for the direct ignition of a blasting charge have been designed. Marshall and other authors describe in detail the manufacture of safety fuses, miner's squibbs, quills, quick match, instantaneous fuse, slow match, detonating fuses, electric fuses, mechanical fuses and shell fuses. The latter are made in two main varieties, viz., percussion and time fuses. The work is generally carried on with great precaution, as regards cleanliness, clothing and safety measures.

Hayhurst in discussing the manufacture of electric fuses refers to noise as a factor in the spinning process of white cotton upon the copper wire to be used for insulation. He refers to the tarring of covered wire as hot and humid work, but considered the hazards in the bridging room, where the soldering of platinum wire across points and running sulphur into moulds around the points was done by girls, as quite negligible.

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Fireworks.—Most of the mixtures used for rockets are practically identical with those used as explosives, such as mealed powder, combined with slow burning substances. Among the different rockets described by Marshall are those used for pyrotechnique displays, the war rocket, employed for incendiary purposes and attacks of air ships. Sound and light rockets, the latter are employed as a flash light to light up positions. Life saving rockets are used by the Coast Life Saving Service and others for throwing life lines. Fireworks also include the various colored lights, such as green, yellow, blue, and red lights. Many of the ingredients such as phosphorus, sulphites of arsenic, antimony and copper are industrial poisons, but their chief danger lies in accidents from explosions. Marshall[63] refers to 24 accidents from 1910–1913 causing 25 deaths and injuries to 64 persons.

Accidents in the Manufacture and Use of Explosives.—Considering the vast quantities of explosives manufactured, the number of accidents is surprisingly small. According to Key[64] during the year 1899 the total quantity of gunpowder, other nitrate mixtures, nitro-compounds and chlorate mixtures manufactured in the United Kingdom exclusive of Government factories was 32,115 tons. There was also produced about 500,000,000 cartridges for small arms, 50,000,000 detonators, electric detonators and fuses and about the same number of yards of safety fuses. In addition there were manufactured about 1788 tons of fire works while the output of fulminate of mercury could not be disclosed.

According to Munroe[65] the output of gunpowder in the United States in 1900 amounted to 10,383,944 lb., blasting powder kegs of 25 lb. each. 8,217,-448 kegs; smokeless powder 7,009,720 lb.; nitroglycerine 51,579,270 lb.; dynamite 130,920,829 lb.; gun cotton 5,905,958 lb. The total output of explosives in the United States in 1905 was 363,748,097 lb., and in 1910 the output was estimated at nearly 500,000,000 lb.

During the decade from 1890 to 1899, 175 accidents occurred in the manufacture of explosives in Great Britain, resulting in the death of 44 persons and injuries to 204 out of a total of 4828 persons employed in the danger buildings, and 11,098 persons exposed in licensed area. This favorable showing is largely due to the intelligence and care taken by the employees and employers and general safety precautions. Among the specially dangerous processes Key[66] enumerates the "milling" or "incorporation" of gunpowder, "the pressing of detonators and the mixing of cap composition."

In addition to the above accidents in actual manufacture 69 accidents with 32 deaths and 81 injuries occurred during the *storage* and *distribution* of the finished product. Among the principal preventive measures Key mentions the following: "(*a*) subdivision of risks, *i.e.*, the number of persons allowed in any one 'danger' building is strictly limited and the communication of explosion between buildings is prevented by the erection of mounds of earth or masonry and by making the quantity of explosive in any building directly dependent on its distance from others; (*b*) scrupulous attention to

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cleanliness; (*c*) prevention of the introduction of matches and other dangerous articles, by providing suitable clothing without pockets and by a thorough system of searching all those employed in danger buildings; and (*d*) the provision of an adequate number of escape doors opening outward, and provided with safety latches so as to yield easily to a push from the inside."

It is the concensus of opinion, that the explosive industry with care and proper safeguard is not an extraordinary dangerous trade, and that the risk in the use of explosives is greater than in its manufacture. This is illustrated in Key's table, showing the number of accidents in the *handling and use* of various explosives during the decade of 1890–99.

Nature of explosive	No. of accidents	No. of persons	
		Killed	Injured
1. Gunpowder*	244	94	294
2. Nitroglycerine compounds	376	135	440
3. Ammonium nitrates	44	12	41
4. Detonators	143	3	193
5. Fireworks	41	19	96
Total	848	263	1064
Total in manufacture during same period	175	44	204

According to Key the majority of these accidents 99 out of 100 could have been avoided by reasonable care. Among the immediate causes of explosions in mines in Great Britain in 1899 which killed 29 and injured 195 were scraping out the detonators with pins, thawing dynamite over the fire, driving dynamite and gunpowder by means of iron rods into roughly drilled holes, and boring out of misfires. Key cites a fatal accident which was reported as follows: A. B. was charging a bore hole in rock with pellet gunpowder. Finding a difficulty in inserting the charge he was holding the tamping rod on the powder while his mate drove it home with a sledge hammer when *for no reason whatever* the charge exploded. The italics are Key's.

It is very evident from the examples of reckless folly cited by Key that much needs to be done in the way of instruction and discipline to diminish this needless sacrifice of life and limbs.

During the year 1913 over 130 men were killed and 250 seriously injured by the use of explosives in the metal mines and quarries in the United States alone. The statistics of the railway bureau for the safe transportation of explosives, cited in 1911 by Professor Holmes in Bulletin 17 Bureau of Mines,

* In the year 1899 alone 146 accidents with gunpowder in mines killed 15 persons and injured 148. Few if any of these were reported, and if multiplied by 10 and added to the casualties due to this explosive, the figures under heading 1 would be enormously increased.

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have shown more than 400 persons were killed or injured and over $3,000,000 worth of property destroyed by explosions in transit by rail.

The losses from the latter accidents in transport have been reduced to almost nothing, and Prof. Holmes declares that the "accidents resulting from the improper use of explosives in mining can be most certainly prevented (1) through the use of the best and safest explosives, (2) through the handling and firing of these explosives in the safest manner by carefully selected and trained men, and (3) through the strict and competent oversight of these men."

Dangers from the Products of Combustion.—Apart from the accidents referred to it should be remembered that the gaseous products of combustion in confined spaces, such as mines and tunnels may prove a very serious danger to the operatives.

The gaseous products in the explosion of various gunpowders averages as follows: Carbon dioxide 49.3; carbon monoxide 12.5; hydrogen 2.2; methane 0.4; nitrogen 32.9; sulphureted hydrogen 2.6 volume per cent. Whenever an explosive is used in which the oxidizing agents are insufficient to convert all the carbon into dioxide, and the hydrogen into water, the amount of monoxide evolved may be greatly increased. The gas from black powder contains between 8 and 17 per cent. of carbon monoxide and many of the explosives used in mines give off more carbon monoxide than dioxide. Marshall[67] refers to several fatalities from this cause in Germany in 1904 and 1908 and it is generally believed that the undue prevalence of respiratory diseases in the South African gold mines and elsewhere is influenced by the presence of monoxide of carbon in these mines. He also states that the high explosives used for military purposes such as gun cotton, picric acid and trinitrotoluene evolve large quantities of carbon monoxide when detonated and hence are unsuitable for underground operations. Sulphureted hydrogen is freely given off by American dynamite. Blown out shots are liable to produce other poisonous gases and vapors such as acrolein and prussic acid. When nitroglycerine is decomposed by simple combustion instead of detonation apart from CO and CO_2 the highly poisonous nitrous fumes are given off. Key[66] refers to a number of instances of poisoning from one, or a combination of these gases. Nitroglycerine explosives according to Jakoby and His, cited by Marshall,[67] distil off part of this constituent which condenses again to a fine mist in the air. If a sweet taste be observed in the air the miners should leave at once and not return until it has disappeared. Dr. Pirrie[68] has recorded his observations on unsuspected dangers to the health of miners from the use of high explosives containing nitroglycerine.

Oliver[69] states that the inhalation of the fumes given off by high explosives may cause violent headache, vomiting, palpitation of the heart and partial or complete collapse. Removal to the fresh air, warm drinks and external artificial heat are the most effective remedies. He also mentions the fact that "many of the miners after the 'gassing' suffer from extreme nervous-

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ness and of a sense of dread attended by a want of confidence in themselves." For symptoms caused by carbon monoxide, carbon dioxide and nitrous fumes. (See list of Industrial Poisons, pages 725, 726, 734.)

EXPLOSIVE HAZARDS IN MANUFACTURING PLANTS, ETC.*

Chemicals, acids and acid vapors:	Hydrochloric acid, hydrofluoric acid, nitric and sulphuric acid.	
Gases:	Benzene, gasoline, naphtha, acetylene, blaugas, gasoline and pintsch gas lighting systems.	
Dust explosions:	Celluloid, coal, cork, dextrin, flour, grains, lampblack, malt, sugar, sulphur, tow, tobacco, wood dust.	
Explosive substances:	Celluloid will explode from percussion and the dust from celluloid will explode from a very small spark of any kind. Chlorates are explosive from slight friction, shocks or when crushed. Collodion is explosive from heat or shock. Dynamite is explosive from heat or shock. Gun cotton is explosive when suddenly warmed or from pressure, shock or friction. Gunpowder is explosive from shock, pressure, friction or heat. Nitrogen chloride will explode under slightest provocation, sunlight or violet sound waves. Nitroglycerine is explosive from heat or shock. Picrates are explosive from heat, friction, shock.	
Liquids which may cause explosions under right conditions:	Acetone	Illuminating oils
	Alcohol	Lacquers
	Benzol	Petroleum
	Carbon disulphide	Spirits
	Ether	Varnishes.
Liquified gases:	Air	Hydrogen
	Acetylene liquid	Hydrogen sulphide
	Ammonia	Oxygen
	Carbonic acid	Sulphurous acid.

INDUSTRIES IN WHICH EXPLOSIVE HAZARDS ARE PRESENT

Bleach, dye, print and finishing departments of textile mills:	Bleaching chemicals: sodium peroxide gives off oxygen and in contact with resin, ethereal oils, or mineral oils, or in a room containing inflammable gases or vapors, may easily cause a fire, or under right conditions, an explosion. Chlorates employed for oxidization purposes especially where aniline black dying is done are liable to liberate oxygen explosively if mixed with potassium cyanide or sulphur, or under the influence of friction, shock, concussion or heat.
Breweries:	Explosions from dust are liable to occur in the cleaning and separating room, also in malt grinding mills, from a foreign body such as a nail, etc., in the grain, striking a spark on the metallic part of the machine.

* Condensed from "Explosive Hazards in Manufacturing Plants" by J. Corbit Barden, Associate Director Bureau of Inspection and Accident Prevention. Published by the Aetna Life Insurance Company, Hartford, Conn., 1914.

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Candy factories:	Dust in starch buck and sifting and drying rooms. Shellac and alcohol also create explosive hazards in these establishments.
Clothing factories:	From benzine cleaning or gasoline gas system for heating irons.
Cotton mills:	Dust and lint, open lights.
Cotton seed oil mills:	Dust and lint should not be allowed to accumulate in these rooms and material should be passed over magnets before going to linters.
Cordage and twine works:	Dust and oily flyings create explosive hazards.
Fertilizer works:	If cooking is done in closed steam kettles, a reducing valve, safety valve, gauge and proper drain should be provided for each kettle.
Flour and cereal mills:	Dust explosions not infrequent.
Garages:	From gasoline, calcium carbide and Presto-Lite tanks.
Glass works:	From fuel oil heating system.
Glucose and starch works:	Dust explosions in grinding rooms and drying rooms, also from sulphur burners and nitric acid used in converting processes.
Grain elevators and grain threshing:	Dust explosions from hot bearings, open lights, static electric sparks or any other form of fire. Sulphur and carbon bisulphide and other volatile and inflammable materials.
Metal workers:	From fuel oil, acids, japan lacquer, gas-heated japan ovens, dip tanks containing volatile oils.
Motion picture films:	See Celluloid.
Oil cloth and linoleum plants:	Dust in the cork grinding room, linseed-oil boiling, compound mixing, explosive vapors.
Packing houses:	Fires spread rapidly on account of grease and oil.
Patent leather and enamel leather works:	Daubs, japan, varnish, oils, lampblack, naphtha. Direct heat and open flames of any kind should never be used or allowed where coating or mixing is done.
Printing, binding, and lithographing:	From gasoline gas or linotype machines, benzine used for type cleaning, collodion in photo-department.
Pulp mills:	Sulphur and sulphur burners, digesters.
Rice mills:	Same as flour mills, sparks are especially dangerous at the attrition machines.
Rubber mills:	From lampblack and oxides used for compounding. From naphtha especially in the churn room, and in the spreading machines, the latter should be properly grounded to prevent static sparks.
Shirt, collar, cuff factories and steam laundries:	From gasoline gas system and for heating irons.
Shoe factories:	From rubber cement and waterproofing material.
Straw and felt hat factories:	From gasoline gas system, also sulphur and sodium peroxide.
Sugar refineries:	Danger from dust explosion. Vacuum boiling kettles should be provided with proper safety valves and gauges.
Tanneries:	Dust hazards in bark mill rooms. Naphtha in degreasing processes.
Tobacco dryers:	Dust explosion risk is due to the greasy and inflammable character of tobacco dust.
Wood workers:	Dust explosion hazards in sandpapering rooms, also varnish dip tanks and from naphtha, oils and varnishes.

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Industrial aniline poisoning, which is just beginning to be known in this country,[70] has already been the cause of numerous cases of sickness among workers in rubber goods; in reclaiming rubber from scrap, in making aniline from benzene and in using certain washes for printing-press rollers. Dr. G. L. Apfelbach in 1913 reported the first two cases of industrial aniline poisoning in this country, one in a press feeder, and one in a dry color factory. Birge in 1914 reported two cases in painters, who were using aniline black paint. Hayhurst in 1915 reported three cases in men employed at rubber mixing mills and among printing pressmen, who used a roller-wash rich in aniline. Luce and Hamilton in their investigation for the Federal Bureau of Labor in 1915, collected several cases in connection with the rubber industry at Akron, Ohio, and since the publication of the Government report have observed several additional cases. The present war has compelled the manufacture of aniline oil in this country, and in one establishment at Akron with a daily output of 2500 pounds, cases of aniline poisoning are sufficiently common for workmen and physicians to speak of the victims as "blue men" or "blue boys."

The monthly review of the U. S. Bureau of Labor Statistics for June, 1916, contains abstracts of the reports of the British committee on the health of munition workers. On page 83 the necessity of protective measures against exposure to lead, tetrachloride of ethane, nitrous fumes, trinitrotoluol, tetryl, fulminate of mercury and certain lubricating and cooling fluids (petroleum products) used in metal turning is pointed out. It is stated that manipulation of tetryl (tetra-nitro-methylaniline) which is an explosive, produces a light dust which may cause troublesome eczema, also headache, drowsiness and lack of appetite. Tetrachloride of ethane is a solvent for acetate of cellulose and is an ingredient of a varnish employed in water proofing the canvas wings and bodies of aeroplanes. Inhalation of its vapor has caused drowsiness, loss of appetite, constipation, pains in the stomach and in more serious cases, jaundice, liver destruction, coma and death. The Committee considers it absolutely necessary for those working with these poisons to maintain good health, the evidence apparently shows that badly nourished workers succumb more readily than others.

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CHAPTER II

THE HIDE, TANNING AND ALLIED INDUSTRIES

(Involving exposure to anthrax and other occupational infectious diseases)

BY GEORGE M. KOBER, M. D., Washington, D. C.

Tanning. Patent Leather. Leather Gloves and Mittens. Brush and Broom Making. Curled Hair. Furriers and Taxidermists. Upholstery, Mattress Making and Feather Work. Preventive Measures.

The handling of hides and skins, apart from being disagreeable and when dry also very dusty work, is dangerous on account of liability to wounds and anthrax infections.

The most common medium for the transmission of anthrax in handling infected skins or fleeces is the dried blood clots adhering to the hair or wool. Indeed so resistant are the spores of anthrax that infections have occurred not only among the men handling the hides and tanners, but also among workers in the boot and shoe, harness and glove industries.

Dr. E. W. Hope,[1] the Medical Officer of Health of Liverpool, presented an interesting account of 60 cases of anthrax studied in that city during a period of 8 years; of these, 36 cases occurred in dock laborers, "and a large proportion of them were infected in the unloading or carting of hides, either loose or in bales."

In 1905 the Medical Inspector[2] reported a case which occurred in the wife of a dock laborer in South London, who had been engaged in unloading foreign hides and evidently carried the germs in his clothing to his house. Another case occurred at Plymouth in a man engaged in unloading bones. Of 261 cases of anthrax reported in Great Britain, between 1899 and 1904, 86 cases occurred in the hide and skin trade; and of these, 21, or 24.4 per cent., proved fatal. In nearly 50 per cent. of the cases the seat of the pustule was in the neck, as against 29.5 per cent. in the wool industry. This difference has been justly attributed to the habit of carrying the hides on the shoulders, and is a strong argument in favor of removal by mechanical means. Dr. Hope[3] cites two cases of anthrax infection in female bag menders; one of the bags had contained wool or hair, and the other, Egyptian cattle bones. Cases have also been reported in persons engaged in mending tarpaulins which had been used to cover wool.

Dr. Hope states that the hides causing infection came from all parts of the world, but chiefly from China and the East Indies. The material causing infection in the case of hair was derived from Russian sources. He also

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refers to the fact that a large number of bales are transhipped to the United States. Since anthrax was extremely rare 30 years ago among the domestic animals of this country, the infections reported are perhaps chiefly due to the importation of imported hides and fleeces. Ravenel[4] reports 12 cases of anthrax among men and 60 in cattle, which occurred in three localities in Pennsylvania during the summer of 1897. All were traced to a tannery handling hides imported from China. The infection in the cattle was believed to be due to feeding on pastures watered by streams contaminated by tannery refuse. Similar instances have been reported in different parts of Europe, and indicate how imported infected hides may be a source of danger to the animal industry in sections heretofore free from anthrax, and also the danger to public health of permitting tannery refuse to be discharged into streams, without preliminary treatment.

Preventive Measures.—In most civilized countries stringent rules have been adopted for the suppression of anthrax and other diseases communicable from animals to man. Among these rules may be mentioned: compulsory notification of the existence of anthrax; isolation and quarantine of the entire herd; prompt killing of the diseased animal and destruction of the carcass by cremation; and thorough disinfection of the stables and other infected premises. The owner is reimbursed for the loss sustained. These rules were primarily enacted in the interest of animal husbandry. The question of anthrax infection, in its relation to man, has been emphasized only in recent years. In some countries, Germany for example, even now no compensation is paid for sheep and goats which perish from this disease, and as a result the farmer is sorely tempted to sell such infected skins to "fell-mongers."

Tanning.—This occupation calls for strength and endurance as many of the processes are laborious, extremely wet, and disagreeable, and are often carried on under insanitary conditions. Indeed, the odors of "beam houses" are so offensive as to be considered a nuisance to the neighborhood. The injurious influence of these odors has, however, been overestimated.

The handling of dried hides and skins involves the inhalation of more or less dust of organic and inorganic origin, fragments of hair, etc. The mortality rates in the U. S. for 1909 show that 15.2 per cent. of all the deaths among tanners were caused by tuberculosis, 14.3 per cent. by heart disease, 8.8 per cent. by pneumonia, and 8.7 per cent. by Bright's disease.

According to Popper,[5] one-sixth of all the deaths are caused by pneumonia and about 40 per cent. from tuberculosis. Diseases of the digestive organs and of the kidneys are not uncommon. Rheumatic and catarrhal affections, on account of exposure to wet and sudden changes, are also quite prevalent. Many of the hides also have sharp cutting edges, which are liable to produce wounds and abrasions and subsequent septic infections. In the handling of lime skins and during the removal of the hair, the workmen not infrequently get short pieces of hair under the finger nails, giving rise to septic conditions.

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As a result of immersion of the hands in caustic lime solutions Prof. Roth[6] refers to a peculiar affection of the skin, characterized by loss of substance and bright red, shining finger tips, called by the French tanners "rossignol" or "pigeon" and by the Germans "Stieglitz." Other skin and nail affections are liable to develop, especially in persons engaged in scraping sheep and rabbit skins and also from the use of arsenic, the chromates and acid solutions. The danger from anthrax infections constitutes an important occupational risk; 63 such cases with 14 deaths occurred in German tanneries in 1903–1904, and Neisser reported 29 cases in 1905, the majority of which were traced to imported hides. Of 255 cases with 45 deaths, tabulated by Holtzmann,[7] which occurred in the German leather industry between 1906–1910, the source of infection was traced in 154 instances to lamb, sheep and goat skins, and in 101 instances to hides from calves, heifers, cows, steers and wild animals. It has long been known that the handling of dried hides is more dangerous, and Dr. Hamer[8] expressed the hope that the wet process of curing would diminish the spread of infection. But because of the increased weight of wet consignments the majority of imported hides are still cured and shipped in a dry state.

A number of the tanneries in the Netherlands, as a result of long experience, have come to the conclusion that the dried hides are more dangerous as a rule, because the countries from which they are shipped pay little or no attention to the prevention of anthrax. They caution especially against the purchase of hides with bayonet cuts, as such perforations are an indication that the skin came from a diseased animal which had been killed for the sake of the hide. They also fear the danger of arsenic-cured hides from China. Lastly, their observations lead them to conclude that hides imported from the United States are the least dangerous, as they came from abattoirs subject to Federal inspection, where none but healthy animals are permitted to be slaughtered.

Tanning and Dressing Processes.—The old method of using tan bark and sumac has been largely supplanted by the use of chemicals. As a preliminary to either process the hides are first soaked in water and afterward in water to which lime, and sometimes disulphide of arsenic, has been added. This causes the hides to swell and facilitates the removal of the fleshy part of the hide, which with the hair is scraped off, either by hand or by suitable machinery.

The de-hairing process is sometimes done in so-called "sweat chambers" under the influence of lime or its sulphide, or of sodium sulphate. The temperature in these chambers is often above 86° and not only attracts flies, but also favors the development of ammonium sulphide and of anthrax spores, if any be present. After this process, the hides are soaked in acidulated water, or a bath containing animal excrement, for the removal of lime.

"When the hides or skins are ready for the actual tanning, they are placed either in a vat containing the old-fashioned tan bark liquor, or into a

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revolving drum known as a "pin wheel," or into a pit supplied with revolving paddles and containing a dilute solution of potassium or sodium dichromate, acidified with hydrochloric or sulphuric acid, and caused to revolve or to be overturned for 7 hours or longer, after which time, if the "pin wheel" is employed, the liquor is drawn off and replaced by an acidified solution of sodium thiosulphate or bisulphite and the revolution is continued several hours longer. If the pit is used, the skins are removed to another, containing the second solution, and kept at rest or overturned for a like period."[9]

During these processes, the hands and arms of the workmen are exposed to the caustic and toxic effects of lime, chromates, etc. Dr. Neisser[10] reports a number of cases of pustular eczema in chrome-tanning workers and 19 cases of chrome ulcers among 300 workers within 1 year. He also reports similar instances in connection with the tanning industry, resulting from the vapors developed in dissolving the chrome in hot water. Protection with long rubber gloves does not always prevent troublesome skin affections. In establishments in which arsenic-cured hides are handled, or realgar or orpiment is used as a chemical agent, there is a certain amount of danger from arsenical poisoning.

There is more or less danger from lead poisoning during the finishing process of alum-tanned leather, which is rubbed with a mixture of talcum and white lead. Cases of lead poisoning have also been observed in men who, while tacking the hides on frames, are in the habit of holding the tinned tacks in their mouth. This habit is also objectionable from the fact that, sometimes, they have been spilled previously on the floor and are picked up and without washing placed in the mouth and may thus be the means of conveying disease germs. In the subsequent processes of leather dressing there is more or less danger from unprotected machinery. In the buffing process, which is characteristic of chrome-tanned leather, and which is done by means of wheels or revolving drums covered with sandpaper, the air of the room is frequently filled with very fine dust, presumably containing chromates. Since aniline dye stuffs and orpiment are used in the manufacture of "fancy leather," we may expect to find aniline and arsenical skin eruptions and even symptoms of systemic poisoning. Benzine is freely used as a solvent for the various dye stuffs. Dr. W. G. Thompson[11] reports a case of multiple neuritis of both arms in a finisher of leather, caused by arsenic which had been used as a tanning agent and was evidently disengaged during the rubbing and polishing process.

Patent Leather.—The manufacture of patent leather is largely a secret process, but sufficient is known to indicate that the workmen are exposed to such industrial poisons as lead, amyl acetate (zapone lacquer), naphtha, and wood alcohol, which are some of the ingredients of a mixture with which the leather is coated. This mixture, after being heated in iron kettles, is applied to the hide, stretched on frames, and evolves fumes which, even employers admit, cannot be withstood by the workmen for any length of time. The

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men generally work stripped to the waist. "In one of the two factories visited the odor of naphtha was noticeable in every department." Naphtha "drunks" were said to be not uncommon among the workers. The minors at work were all Italians, young and rugged, recently landed, so that the occupation had not as yet affected them.[12]

Preventive Measures.—It is needless to insist that this occupation is intrinsically dangerous to health and that the utmost precautions should be exercised to safeguard, by adequate exhaust ventilation, the health of the employees.

The Leather Glove and Mitten Industry.—The average number of persons employed in this industry in this country during the year 1909 was 12,950, of whom 48 per cent. were males and 52 per cent. females. The average number of children under 16 years of age was 269. The working hours averaged between 54 and 60 hours per week.[13]

The industry involves exposure to dust and toxic dyes and agents used in the preparation of the leather. The work is often carried on under unfavorable environments. Diseases of the respiratory organs, caused by exposure to bad air and dust, especially in the buffing process, are most frequent, and tuberculosis is responsible for 60 per cent. of all the deaths. Catarrhal affections, conjunctivitis, diseases of the digestive organs, varicose veins and leg ulcers are not uncommon. Occasional cases of lead poisoning, from the use of talcum and white lead, and isolated instances of anthrax infection have been reported. Persons engaged in the dyeing process not infrequently develop chrome ulcers on the thumb of the left hand and on the second and third fingers of the right hand. Another form of ulceration has been observed between the toes of employees, who are in the habit of using their bare feet for the purpose of soaking the leather in tubs. The alum used for tanning purposes and failure to wash and dry the feet properly is believed by Bauer[14] to be the cause of these ulcers. Callosities of the hands and contracted tendons are also observed.

Preventive Measures—All home work should be discouraged and the industry should be carried on in sanitary shops properly quipped with exhaust ventilation for the removal of dust and vapors.

Brush and Broom Making.—In the manufacture of brushes, hog bristles and vegetable fiber are chiefly used; for the more delicate brushes, horsehair, goat, camel and cattle hair, and hog wool are employed. The handling of these hairs in a raw state is a dirty, dusty and even a dangerous process, on account of the possibility of anthrax infection.

Of the 60 cases of anthrax infection reported by Dr. Hope in 1912, 7 contracted the disease in handling horsehair or bristles in hair factories; 14 cases with 7 deaths occurred in German hair and brush factories in 1904.[17] It is usual and necessary, therefore, to boil the bristles for several hours, after which they are dried in steam dryers and bleached, and then dressed or combed by machinery and mixed. In the manufacture of paint, varnish

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and kalsomine brushes, the bristles are set into the handles by means of a tar cement or a cement composed of wood alcohol and shellac; for shaving brushes, a cement of rosin and linseed-oil is used, which is kept fluid by means of electric or non-luminous gas flame heaters, provided with hoods. Hirt reports that nearly one-half of all the deaths among brush makers are caused by consumption. The danger is probably due to the irritation produced by the inhalation of sharp fragments of bristles, which are not easily dislodged. Instances of lead poisoning have also been reported in this industry.

In the nine factories reported upon by the Massachusetts State Board of Health[18] "the dust removal appliances were found to be fairly good, satisfactory and even excellent," and the health of the employees, mostly women and girls, "appeared to be fair or good."

When vegetable fiber is used, dust production is also very great. This is especially true in the manufacture of "corn brooms." This danger is recognized, so that hooded stripping machines, provided with a chamber for the removal of dust, seeds, and other foreign material, have been installed. In some establishments the "wet process" has been adopted before removal of the seeds and dust. The bleaching process involves exposure to sulphur and chlorine fumes; aniline solutions are used for dyeing and staining purposes. Fatigue from piecework, long hours, jarring processes, faulty position and unfavorable working conditions are among the occupational hazards observed in this industry.

Curled Hair and Horse-hair Cloth Factories.—In all of these industries, apart from the inhalations of mixed dust and of minute fragments of hair, there is danger of anthrax infection and other communicable diseases. Of the 261 cases of anthrax reported in Great Britain between 1899 and 1904, 70 cases occurred in the hair industry. Nichols[18] of this country reported 26 cases, which occurred in one curled hair factory in 3 years. The Massachusetts State Board of Health for 1908 reports four cases of malignant pustule, in a curled hair shop, during a period of 7 years. Since the management adopted a method of disinfection, by prolonged fumigation with formaldehyde and subsequent boiling of the hair, no cases have occurred. A peculiar painful affection of the finger tips, extending at times to the margin of the finger nails, has been observed in some of the employees in hair cloth factories in France.

Furriers and Taxidermists.—A similar affection, but more especially limited to the finger nails of men engaged in scraping rabbit and hare skins, was reported by Dr. Glibert of Brussels in 1896. It was noticed in 18 of the 22 workmen examined by him. The character and amount of dust evolved in the various processes of the fur industry are doubtless factors in the development of respiratory diseases. This is especially true in the manufacture of artificial furs and the preparation of rabbit fur for the felt hat industry. But in the manipulation of all kinds of fur, there is more or

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less dust inhalation, incident to repeated brushing, fur pulling and clipping, and such dust not infrequently harbors disease germs. Dr. W. G. Thompson[19] states that pulmonary abscess is very common among the employees of a fur-manufacturing establishment in the State of New York. Arsenious acid in the proportion of 1 lb. to the gallon of water is often employed by furriers and taxidermists as a preservative. An American chemist demonstrated its presence in 11 of the 42 samples examined, amounting in some instances to 170 grains per square yard. Many of the furs are in whole or in part dyed with lead pigments, which constitutes an additional source of danger.

A clinical and sanitary study of the fur and hatter's fur trade has recently been conducted by the Department of Public Health in the City of New York (see Monthly Bulletin for October, 1915).

Of 3839 persons engaged in dressing, dyeing or manufacturing fur, a complete physical examination was made of 542 persons. Of these, 67 individuals suffered from anæmia and 18 others showed marked malnutrition; 12 persons suffered from furrier's asthma; 32 from subacute and chronic bronchitis; 11 from tuberculosis and 7 from emphysema; 163 suffered from skin diseases; 151 from nose and throat affections; 74 from diseases of the heart; 89 from ocular defects; 50 from dental defects; and 35 from orthopedic defects, inclusive of 14 cases of varicose veins and 10 of flat-foot.

A number of the dyers and dressers showed the presence of dye pigments and sawdust in their nostrils. The nails of dyers frequently revealed deformities. Callus formations on the fingers, hands and on the flexor surface of the wrists were not uncommon in cutters, dressers and beaters. Only 77 individuals of those examined (about 14 per cent.) were found free from physical defects.

Preventive Measures.—Reference has already been made to the danger of mercurial poisoning involved in the manufacture of felt hats. It is needless to insist that adequate exhaust ventilation should be provided. It is gratifying to note that the morbidity in establishments which have provided such safeguards has been reduced fully one-half.

Upholstery, Mattress Making and Feather Work.—These occupations have always been regarded as unhealthful, on account of the inhalation of dust incident to the manipulation of hair, wool, cotton, felt, excelsior, feathers, and other material. Even when new material is used, unless it has been thoroughly washed and disinfected, the process of picking and carding involves the inhalation of large amounts of dust which may contain disease germs, and which predisposes to respiratory diseases, anthrax infections, etc. All these dangers are enormously increased when second-hand material is handled, as in the renovation of mattresses, feather beds, bolsters, pillows, furniture cushions, etc., especially when such articles have been used by infectious patients.

It is not surprising that a French Commission should have found that

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"old mattresses are often the means of spreading the germs or virus of smallpox, erysipelas, scarlet fever, diphtheria, measles and typhoid fever." As a matter of fact, among the few cases of smallpox reported in Germany in 1905[20] several were traced to a feather bed renovating establishment at Pilsen. The dangers of handling even new feathers imported from Russia, China and countries where smallpox prevails have been pointed out by Shablowsky.[21] In the opinion of Sir Thomas Oliver[22] feather dust is liable to produce ophthalmia and diseases of the respiratory organs.

Preventive Measures.—Fortunately in most of these occupations hooded machines with exhaust ventilation have taken the place of hand labor in the picking and carding processes of hair, wool and other material; but in spite of dust-removing devices, more or less dust escapes, and every effort should be made to render them more effective. A more general adoption of the wet processes will be a great aid. Even if the dust is free from disease germs, the inhalation of minute fragments of hair and organic and inorganic matter, some of which is derived from the perspiration of animals which has dried on the hair of horses, etc., doubtless predisposes to diseases of the respiratory system. It is well known that cavalry troops are more susceptible to pneumonia than other arms of the service. The writer in 1886 pointed out that the inhalation of organic dust, evolved in the grooming of horses, was a possible predisposing factor.

There is no question that old as well as new material of animal origin should first be subjected to disinfection, by means of steam under pressure, by formaldehyde, or by boiling. Old and worthless bedding, used by infectious patients, should be burned. The precautions for the prevention of anthrax, especially in the handling of imported hides, hair and wool, have been described on page 572, but as it is extremely difficult to kill the spores of anthrax, it is desirable that the workers should be informed of the dangers, and their coöperation secured in the prevention of this industrial infection. Wounds and abrasions, pimples and skin affections should receive prompt attention. (See also page 158.)

A most praiseworthy example has been set by the Pachetti Bros., owners of a hair-cloth factory in Milan in which 700 persons are employed. Although the horsehair is disinfected by steam under pressure, a physician makes daily visits, treats all wounds and abrasions, and examines all cases of indisposition, with special reference to anthrax infection. His office is equipped with apparatus for a bacteriological diagnosis and the prompt administration of Sclavo's serum.

Robinson and Wilson[23] refer to the occupational hazards in the the *leather industry* in Cincinnati. Among 197 men employed in the manufacture of leather, 3 or 1.5 per cent. were found to be tuberculous. In one of these cases the occupation was in no way related to the immediate cause of the disease. The low incidence of tuberculosis among a class of workers exposed to many deleterious influences is attributed largely to the selection of strong able-bodied men, sufficient air and floor space, favorable working conditions as regards temperature and humidity after the hides have left the vat, and the fact that over

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50 per cent. of the men examined were more than 35 years old and hence beyond the susceptible age.

Among 57 men engaged in the manufacture of *harness* and *saddles*, 3 or 5.2 per cent. were found to be tuberculous. The investigators consider the number examined too small to hold the industry responsible for the high percentage, since with the exception of the collar-making department, there is little exposure to dust. The collars are stuffed with rye straw and chaff, and the operations are attended with much dust, but the blowers connected with the cutting and stuffing machines carried away most of the dust. Moreover none of the tuberculous subjects were working in this department.

All of the 14 workers employed in two *broom factories* were found to be in good health and presented no evidence of tuberculosis, in spite of poor light and ventilation, and promiscuous spitting. Of 48 persons engaged in the manufacture of *feather pillows*, *cushions* and *bedding goods* only one tuberculous subject was found. No history obtainable. All dust producing machines were supplied with exhaust fans.

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CHAPTER III

THE GOLD, SILVER, JEWELRY AND ALLIED INDUSTRIES

BY GEORGE M. KOBER, M. D., Washington, D. C.

Gold Mining and Extraction. Goldsmiths. Cheap Jewelry and Electroplating. Silversmiths. Manufacture of Watches and Clocks. Leaf Metal Workers. Nickel and Nickel Plating. Iron Carbonyl. Preventive Measures.

Gold Mining.—The dangers of gold mining do not differ from those of metalliferous mines in general; indeed they are probably less in alluvial and placer mines. In quartz mines, however, the men are exposed to a very irritant dust as shown by an alarming prevalence of pulmonary diseases, especially in the gold fields of the Transvaal and at Bendigo. The dust is generated not only during the drilling and blasting of the hard quartzite rock, but the men are exposed also during the transport of the conglomerate to the surface and the subsequent crushing in the stamp mills. (See dust prevention, page 440.)

Extraction of Gold.—After repeated washing of the crushed ore the gold is extracted by amalgamation with mercury, which takes up the gold and from which it is recovered by raising the temperature to 400°C. This involves danger from mercurial poisoning, especially during the retorting process.

The cyanide of potassium process is now generally employed as a substitute for the more dangerous mercurial process. In this process the crushed ore is placed in vats containing a 0.05 to 0.5 per cent. solution of cyanide of potassium, where it remains from 12 to 24 hours or longer according to the character of the ore. The liquor is then passed over pure metallic zinc shaving, upon which the gold is deposited as a slime. This slime is roasted and smelted and pure gold is obtained. Work with the cyanide solution, however, involves exposure to cyanogen vapors unless guarded against by copious ventilation, and also necessitates the use of rubber gloves in order to avoid troublesome eczema of the hands and arms.

Silver is likewise recovered from crushed ore by amalgamation with mercury, and considerable quantities are obtained from lead ore during the roasting of which there is a certain amount of danger from lead, arsenic and antimony poisoning. (See lead smelting, page 78.)

Gold and silver apparently possess no serious toxic properties. Cases of argyria, *i.e.*, a brownish-black discoloration of the skin as a result of ingestion or penetration of silver dust, have been reported in silver workers. Koelsch[1] describes two cases in silver leaf workers. Most of the cases have been observed among workers in the glass-pearl industry, who employ a solution of

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nitrate of silver for silvering purposes and apply suction by the mouth. This danger has been obviated by the use of air pumps. Cases of local argyria have been observed in photographers and mirror platers.

Goldsmiths.—The manufacture of jewelry affords employment to a large number of persons, including females and minors; of 5753 persons employed in various jewelry establishments in Massachusetts[2] there were 3504 males and 2219 females, including 442 girls and 260 boys. The Sanitary Inspector found, what is quite generally the case, a tendency to overcrowding; this together with a stooped position over bench work, in the absence of adjustable seats, and a high temperature of the room constitute in themselves injurious factors, aggravated by dust, acid fumes and other conditions, such as fatigue due to eye strain incident to the various processes which will be briefly alluded to. The mortality rate from diseases of the lungs in the Berlin goldsmiths[3] from 1886–1893 was 42.5 per cent., inclusive of 40.2 per cent. from tuberculosis.

The basis for all kinds of jewelry is what is known as "flat stock," which is made by sweating on bars of gold by means of silver solder to bars of brass of the same width in a gas furnace to a cherry red which results in a firm fusion. This process is necessarily attended with exposure to excessive heat and should never be carried on in the general workroom. The bars are then rolled to any desired thickness, the gold spreading equally with the brass, and from this "flat stock" both tubing and wire may be made suitable for bracelets, slides, watch chains and ornaments of every description. The patterns for the flat ware are struck by means of a steel die and cut out in steel presses. The several parts are then soldered together usually by female labor; the process involves the use of Bunsen burners and blowpipes, and although an ingenious substitute for the blowpipe operated by compressed air has been employed in Germany, the work which is done within 16 in. from the eyes, involves not only eye strain, but also exposure to heat, inhalation of the products of combustion, and possibly of coal gas from defective rubber tubings and burners. After soldering, the piece is placed in a 5 to 20 per cent. solution of sulphuric acid for the removal of borax and is then polished with cotton cloth wheels and bristle brushes. The polishing wheels are provided with efficient hoods and exhausts so that the dust may be collected under water in suitable tanks for the ultimate recovery of the gold and silver. The final finish consists in the application of a weak solution of potassium cyanide and gold; this is applied steaming hot in open crocks or sinks, not infrequently located in the center of the room.

Cheap Jewelry and Electroplating.—The manufacture of cheap jewelry, ornaments, etc., is a growing industry both here and abroad; suffice it to say that most of these trinkets are merely plated brass goods, which are first cleaned by means of an acid bath containing nitric or muriatic acid, although sulphuric acid is sometimes used. This dipping process in hot acid solutions, apart from being a frequent cause of eczema of the hands and arms, also

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exposes the workmen to injurious fumes. The goods are then washed off in ammonia solutions and dipped in the electroplating bath consisting usually of gold or silver dissolved in a solution of potassium cyanide, the fumes of which constitute the chief element of danger. (See also copper, nickel, tin and zinc plating.) Occasional cases of lead and mercurial poisoning have been observed in galvano-plastic operations and also in the refinement of gold and silver.

Plated goods are not polished but they are subjected either to the so-called scratch brushing process, or various enamels and lacquers are employed to prevent tarnishing. Zapone lacquer is quite generally used and the observations of employees of the State Board of Health in Massachusetts as to its effects upon the health of some of the female employees indicate the wisdom of placing it in the list of industrial poisons. (See Amyl Acetate, page 721.)

The machinery used in the jewelry industry includes power presses, foot presses, stamps, rolling mills, large and small lathes, milling and grinding machines, planers, grindstones, small sand blasts, emery and cotton wheels. Occasional injuries to the fingers occur in operating the stamp and pressing machines. Dr. Baum,[4] a female Medical Inspector, has called attention to the fact that miscarriages and premature births are quite frequent among the women employed in the Pforzheim jewelry industry and attributes this largely to the use of the foot power presses. The writer feels disposed to raise the question whether the constant handling of brass goods may not cause a subtle form of lead poisoning to which the embryo is peculiarly susceptible.

The materials employed in the manufacture of jewelry besides those mentioned also includes aluminum, precious and imitation jewels, celluloid, glass and pearls; in fact quite a number of establishments are also engaged in the manufcture of pearl goods.

Silversmiths.—The hygiene of silversmithing does not essentially differ from the industry just described; both occupations are sought by weaklings and the working conditions tend to produce not only pulmonary diseases but also visual defects, because much of the work is done at close range and unless the light is sufficiently ample, eye strain results. On the other hand, if the light is too glaring, or the persons handle highly reflecting surfaces, irritation and hyperæsthesia of the retina is apt to develop. This condition is by no means uncommon in polishers of silver or silver-plated ware.

Preventive Measures.—The most injurious factors in these industries are exposure to vitiated and often overheated air, dust of a mixed character, acid fumes, the vapors of cyanogen compounds, ammonia, zapone lacquer and of glacial acetic acid, while the odor of sour beer, solutions of soda, bran and shorts and soap tree bark employed in the "scratch-brushing" process are also more or less offensive. All these dangers can be reduced to a mini-

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mum by efficient exhaust ventilation, separate rooms for the "dipping" department, provided with suitable hoods and suction apparatus. The persons engaged in the "dipping process" should either handle the goods with wooden tongs or protect their hands with zinc ointment or rubber gloves in order to avoid troublesome affections of the skin.

Manufacture of Watches and Clocks.—In this industry there is likewise a tendency to overcrowding, and failure to provide adjustable seats for bench work. The lighting of the rooms is often defective, causing eye strain in about 10 per cent. of those engaged in the work (Cohn).

The injurious factors, apart from exposure to dust derived from gold, silver, steel, copper, brass and the material used for enamel, are the fumes of nitric acid evolved in the enameling process of dials, etc. The latter process and the tempering of watch and clock springs, and the casting of leaden weights also involve exposure to lead. The fumes of benzine and of cyanide evolved in cleaning the small parts are likewise inimical to health. The Berlin statistics[5] from 1892–97 show that 50 per cent. of all the deaths in this industry were caused by tuberculosis and the average age was only 27.8 years. This may in part be accounted for by the fact that many persons with feeble physique enter this industry, but the bad working conditions are also important contributory factors.

Preventive Measures.—Most commendable efforts are made in modern establishments to provide proper safeguards in the way of air-space, light and ventilation, with mechanical devices for the arrest and removal of dust and fumes at the point of their origin. In one of the German establishments the tempering furnaces are supplied not only with exhaust ventilation but also with a pyrometer, in order that the operators can observe the color of the steel without exposure to the vapors.

Leaf Metal Workers.—The metal employed for gold leaf, silver, bronze, aluminum, or other composition, usually an alloy of copper and zinc, is first rolled into thin sheets and subsequently hammered into the finest sheets inclosed in parchment paper. The hammers weigh from 6 to 24 lb. and the workmen lift this weight about 90 times every minute, which involves a hard day's work. Recently special beating machines have been invented which it is hoped will obviate excessive muscular efforts, exposure to intensive noises and accidents from hammer blows.

Leaf metal is employed in the manufacture of mouldings, picture frames, gilding and silvering of furniture, art goods, etc. The leaf metal or other gilding materials are generally applied by means of a size containing benzol or benzine, acetone, pyroxylin, wood alcohol, amyl acetate and ammonia. Apart from exposure to these agents and leaf metal dust, more or less white lead and acetate of lead are used in this industry and add an element of danger. The work is generally done in closed rooms to avoid draughts and hence the air is charged with the odors of glue, varnish, oil, turpentine, shellac and the other agents mentioned. In the manufacture

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of gold-threaded textiles, a very fine metallic dust[6] is evolved which has been known to produce catarrhal conditions of the eyes.

According to Hirt,[7] the leaf-metal workers are peculiarly liable to diseases of the respiratory organs and especially to tuberculosis.

The gold leaf workers in Vienna[7] have a very high morbidity and mortality from chronic bronchitis, emphysema, and tuberculosis of the lungs. The morbidity of this class of workers in Austria, according to Jehle,[8] is 42.5 per cent. against an average of 27.5 per cent. in other pursuits.

The vital statistics of *leaf metal workers* in this country are extremely meager. Hoffman's statistics are based upon 25 deaths from all causes; of this number 7 were from consumption and 5 from pneumonia and other diseases of the lungs.

Nickel.—This metal which has come into general use was believed to be free from toxic properties. When derived from arsenical ore, there is danger from arsenical fumes during the roasting operations. Mond in 1890 discovered that by passing a current of carbon monoxide over finely divided metallic nickel, a gaseous compound of nickel and CO was formed. When heated to 150°F. the gas is decomposed into its constituents and metallic nickel is deposited.

Nickel carbonyl ($NiCO_4$) is a clear, pale, straw-colored liquid which boils at a temperature of 43°C. This salt is quite volatile and a peculiar soot-like color is perceptible when present in the air to the extent of 12,000,000 vols., while a Bunsen flame becomes luminous when it is present to the extent of 1,400,000 vols. These two characteristics are relied upon to detect the escape of the gas during the manufacture of nickel carbonyl, which, on account of its toxic properties, must be carried on in hermteically closed iron chambers.

The process was introduced in the beginning of the present century and about 25 workmen were poisoned, some fatally, before the danger was fully recognized. In the milder forms of poisoning the men suffer from headache, giddiness, unsteady gait, nausea and at times dyspnœa, which symptoms quickly disappear upon removal to fresh air. In the more severe forms, the dyspnœa, after a lapse of 12 to 36 hours, is increased, accompanied by cyanosis, a rise in temperature, and cough, with more or less blood-stained expectoration. The action of the heart is usually found to be increased, but otherwise normal. Delirium of varying types is generally present. According to H. W. Armit,[9] the fatal cases terminated between the fourth and eleventh days. Post-mortem examination revealed hemorrhages in the lungs, œdema of the lungs, and hemorrhages in the white matter of the brain. Some doubt exists as to whether any blood changes were present. Oliver states that some of the deaths were almost instantaneous. In the case of one man who died after 3 days' illness the lungs were found to be œdematous and intensely congested. In another case there was inflammation and consolidation of the lungs. In the third case, who died on the eighth day, numerous hemorrhages were found in the brain and cerebellum and the nerve cells of the

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respiratory nucleus in the medulla oblongata showed distinct chromolytic changes. For some years it was believed that these cases were instances of carbon monoxide poisoning.

Armit experimented upon animals to determine: (1) whether the symptoms and lesions observed in nickel carbonyl poisoning were caused wholly or in part by the CO of the compound; (2) whether nickel carbonyl is absorbed as such; and (3) whether the nickel of the compound is the toxic factor. He concluded that the poisonous effects of this compound are due to nickel and not to carbon monoxide, and thinks the peculiar toxicity is caused by a deposit of nickel, as a slightly soluble compound in a very fine state of subdivision, over the immense area of the respiratory surface, where it is dissolved by the tissue fluids and is then taken up by the blood. The hemorrhages found after death are in his opinion caused by fatty degeneration of the vessel walls, the specific pathological change having been set up by the nickel.

Iron Carbonyl.—A light sherry-colored liquid which according to Oliver[10] is, like nickel carbonyl, extremely volatile and equally poisonous.

Preventive Measures.—As cases of nickel carbonyl poisoning occurred only when hand labor was used, during a breakdown of the automatic machinery, it is important that the utmost precautions for the detection and removal of the gas be taken.

Nickel Plating.—The men engaged in nickel plating not infrequently develop a peculiar, eczematous, vesicular inflammation of the skin affecting the hands and arms and occasionally also other portions of the body. The disease is attributed by some authors to the caustic action of nickel salts, especially nickel sulphate or nickel cyanide, employed in the electrolytic bath. Others hold that such agents as benzene, petroleum, soap, and lime, which are quite generally used in all plating processes, are responsible for the mischief, while still others maintain that it is the result of a combined action of various irritants. Whatever the exact cause may be, careful attention to the skin, in the way of cleanliness and the application of vaseline or cold cream, are indicated as protective measures.

Surgeons D. E. Robinson and J. G. Wilson[11] report that in an examination of 100 workers in the manufacture of *Jewelry* in Cincinnati, only one case of tuberculosis was found.

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CHAPTER IV

IRON, STEEL AND ALLIED INDUSTRIES

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Blast Furnaces. Bessemer Steel Works. Electric Welding. Puddling. Mechanica Departments. Prevention of Accidents. Foundries. Core and Mould Makers. Cleaning and Milling Castings. Blacksmiths, Forgemen, Boiler Makers, Riveters, Structural Iron Workers, etc. Cutlery and Tool Industry. Tempering. File Cutting. Gunsmiths, Fire Arms, Weapons. Agricultural Implements. Iron Sanitary Ware Factories. Wire Industry.—Wire Cloth, Netting and Fencing Material. Tin Industry—Tin Plating, Tinners, Tinsmiths. Vanadium. Preventive Measures.

Blast Furnaces.—The operation of reducing iron ore in a blast furnace is necessarily continuous and most furnaces operate with two 12-hour shifts and work 7 days a week. During the year 1909, of the total number of wage earners 31,729, or 82.6 per cent., were employed in establishments where the prevailing hours were over 72 hours a week, and only one-half of 1 per cent. worked between 54 and 60 hours per week. The operatives, apart from hard work, are exposed to excessive heat, abrupt changes in temperature, to all kinds of injuries, burns and explosions, more or less dust and sand and the inhalation of toxic gases from leaky furnaces, gas valves or mains which especially endanger the men while engaged in cleaning out the furnaces or during the process of tapping the slag or the metal. The furnace gases contain carbon monoxide and dioxide in large quantities, compounds of sulphur and cyanogen and even lead fumes, if lead happens to be present in the iron ore. In granulating the slag sulphureted hydrogen is also evolved.

Accident Liability.—Of 19,604 blast-furnace "300-day workers" reported for the year ending June 30, 1910, by the U. S. Department of Labor,[1] there were 5047 accidents, a rate of 257.4 per 1000; of these, 60 or 3.06 per cent. proved fatal; 50 or 2.55 per cent. resulted in permanent and 4937 in temporary disability.

Preventive Measures.—Great care should be taken to prevent the escape of poisonous gases and much has been done toward their utilization for heat, power and illuminating purposes in the steel plants, and commendable efforts are being made in safeguarding the operatives against accidents, explosions, etc., by the construction of modern strong furnaces with water-cooling jackets and improved methods in the stoking and charging of the various materials and in the tapping, pouring and transportation processes.

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Bessemer Steel Works.—In the conversion of pig iron into steel by the Bessemer process, apart from the hard work and exposure to intensive radiant heat and escape of gases, the operatives are exposed to serious injuries from falling scraps from above. Burns are most frequently caused by flying sparks of "molten metal" from the converter during a blow, from spilling of the hot metal ladles, and from explosions on account of the presence of moisture in the converter or from the normally occluded gases of the molten metal.

FIG. 37.—Welding a steel ring with an electric arc. The workman's eyes are protected from the blinding glare by three panes of colored glass in the helmet, which also defends his face and neck from the rays that destroy the skin. (Photo suppied by Würdemann, taken from *The World's Work.*)

Electric Welding.—In the electric welding of steel, etc., the light produced is extremely intense (equal to about 8000 candle-power) and, as the operation has to be closely observed, the workmen must protect their eyesight by shields composed of six alternating layers of blue and red glass. The light and heat evolved are sufficiently intense to cause a condition of the skin comparable to sunburn.

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Hirschberg[2] describes a condition of the eyes in persons exposed to strong electric light, especially in the electric welding of steel characterized by lesions of the conjunctiva, cornea and retina and designated by him as "electrical ophthalmia." Crzellitzer[3] has reported similar cases.

Dr. Apfelbach[4] cites 50 cases observed by him among workers of the Illinois steel plants, caused by electric light, redundant with ultra-violet rays, causing conjunctivitis, photophobia, profuse lacrymation, temporary loss of vision, and rarely keratitis, retinitis, permanent pigment changes of the retina and scotomata.

Accident Liability.—Of 3668 300-day workers in the Bessemer department of steel plants reported by the Bureau of Labor there were 1552 accidents or 423.1 per 1000 with 16 deaths, or 4.36 per 1000; 11 or 3 per 1000 resulted in permanent disability and 1525 in temporary disability.

Open-hearth Steel Works.—The temperature conditions under which the men work during particular operations have been studied by the Bureau of Labor.[5] In spite of the fact that all doors and ports were water cooled and furnace fronts partly cooled, the thermometer 6 or 7 ft. in front of an open hearth with the door partly open, during the operation of rabbling the bottom and stirring heat, registered 120° and 155°; at a distance of 2 ft., and while banking doors with dolomite, the workmen were exposed for 2 minutes at least to a temperature of 220° and over. The excessive heat and exposure to intense light in the various processes of steel making and in electric welding must be observed in order to be fully appreciated, and naturally calls for special protection of the eyes by means of suitable helmets and smoke-colored glasses.

Accident Liability.—Of 9017 employees, 3067 were injured—an accident rate of 32.6 per 1000 with 30 deaths (3.33 per 1000); 38 or 4.21 per 1000 resulted in permanent and 2999 or 4.21 per 1000 resulted in temporary disability.

Puddling.—The process of puddling, the oldest of the purifying methods by which iron is produced, is carried on in a furnace supplied with a large fire-box on one end, the flames of which are reflected upon the pig iron placed in an adjacent chamber. When the mass begins to liquefy, the puddler by means of a rabble (a hoe-shaped iron) stirs the molten metal and as the impurities are burned out the melting point rises and the metal solidifies into smaller masses, which are then gathered into two or three huge balls. They are then removed and hammered or squeezed for the removal of slag. The work is extremely laborious and attended by exposure to intense heat and often reckless exposure to cold draughts. Apart from injuries to the eyes, those exposed to the intense heat and light are liable to suffer not only from chronic conjunctivitis but also from more serious affections of the eye, which may result in retinal and choroidal lesions and also in cataract. Dr. de Schweinitz, cited by Edsall, states that he could often tell whether iron puddlers were right handed or left handed by studying the effect of their work

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on their eye grounds. Quint relates cases of right-sided cataracts in right-handed iron workers and cataracts of the left eye in those who were left-handed. It is very likely that exposure to intense heat causes dehydrization of the lense and other pathological conditions.

Many of the employees have intemperate habits and as a result of combined influences often develop diseases of the respiratory and circulatory system and the kidneys.

Accident Liability.—The liability of puddlers to accidents is not very great; of 1239 workers investigated by the Bureau of Labor there were 62 injuries; none proved fatal; most of the accidents consisted of minor burns and sparks flying into the eyes producing only temporary disabilities.

Rolling Mills.—In this department the men are likewise exposed to excessive heat, hard work, to burns from flying hot metal and slag and the various injuries incident to the moving of large masses. In the hand-operated mills the risks from burns and similar injuries incurred in handling the heated billets and managing the material with tongs and hooks are very much greater than when mechanical means are employed; on the other hand, the fatality rates and permanent injury rates are greater in mechanical mills, simply because if a man is caught in moving machinery a severe or fatal injury is likely to result.

Accident Liability.—In 13,566 workers in mechanically operated mills there were 4199 accidents or 309.5 per 1000; of these, 28 or 2.06 per 1000 proved fatal; 40 or 2.95 per 1000 resulted in permanent and 4131 in temporary disability. Among 10,675 workers in "hand-operated mills" there were 3917 accidents or 366.4 per 1000; of these only 11 or 1.03 per 1000 proved fatal and 28 or 2.62 per 1000 resulted in permanent disability.

Mechanical Departments.—All steel plants employ a large number of mechanics covering a wide range of occupations. Those engaged in repair work are liable not only to accidents incident to their own occupation, but also those occasioned by the work of others, such as unexpected starting of machinery; leakage of gas where the work is being done also frequently adds to these special hazards.

Accident Liability.—Among 17,421 workers reported by the Bureau of Labor there were 4147 accidents or 238 per 1000; of these, 23 or 1.32 per 1000 proved fatal, 31 or 1.78 per 1000 resulted in permanent injury and 4093 in temporary disability.

Preventive Measures.—The dangerous machines in shops are the saws, laths, drills, planers, etc., and it is extremely gratifying to note that the accident rate in the mechanical department of one of our large steel plants has been reduced from 388 in 1905 to 114 in 1910 as a result of effective safety devices.

Yards.—Among 16,441 employees in steel plant yards reported upon by the Bureau of Labor there were 2483 accidents or 151 per 1000; of these, 47

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or 2.86 per 1000 proved fatal and 23 or 1.40 per 1000 resulted in permanent injury and 2413 in temporary disability.

Summary of Accidents.—The investigation by the Bureau of Labor,[6] from the report of which the foregoing data have been compiled, covered all the accidents which occurred during the year ending June 30, 1910, in 155 steel plants representing a working force of 158,604 employees, equivalent to 146,979 300-day workers. The general accident rate was 245.2 per 1000;

FIG. 38.—Hot saw in beam mill thoroughly guarded to prevent sparks from flying through the mill, to the injury of the workmen. (Photo supplied by Würdemann through the courtesy of Carnegie Steel Company.)

the fatality rate was 1.86; the rate resulting in permanent injury was 2.72; and the rate resulting in temporary disability of 1 day or over was 240.6 per 1000. In brief, although nearly one-fourth of the employees met with injuries, only 1.9 per cent. of all the accidents resulted fatally or in permanent injury.

A very interesting table on page 69 of the same report, covering 130 plants for 2 years, ending June 30,1910, and one plant for 6 years, ending December 31, 1910, specifies the nature of the disabling injuries, number of days lost, etc. Of the 18,959 cases thus tabulated, asphyxia occurred in 710 or 6 per cent. of the cases; fractures, 497 or 2.6 per cent.; dislocations, 555 or 2.9 per cent.; injuries to the eyes, 1056 or 5.6 per cent.; burns, 2842 or 15 per cent.;

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injuries to the foot, 3306 or 17.4 per cent.; injuries to the hand, 4908 or 25.9 per cent.; injuries to other parts, 3968 or 20.9 per cent.; all other injuries, 1716 or 9 per cent. It is a remarkable fact that the percentage of injuries to the eyes according to the Imperial Insurance Office in 1897 was about the same in the German iron and steel industry. Two-thirds of the eye injuries were caused by foreign bodies and the remainder by flying sparks.

The report emphasizes the fact that for a large group of plants in which the work of organized accident prevention is well developed the accident rate was found to be as low as 167 per 1000, while on the other hand in groups of plants in which safety work has not yet been developed it was as high as 506.9 per 1000, and in some of the individual plants in this group considerably higher.

According to Röpke,[7] during 1909 among 165,470 insured employees in the "Rhein" steel plants of Germany 27,895 accidents were reported or 169 per 1000 workers; of these, 183 resulted fatally, 198 in total and 1630 in partial permanent disability, and 513 in temporary disability. It should be stated, however, that according to the Report of the Bureau of Labor the German accident figures include no temporary disabilities of 13 weeks and under. According to the same report, more than half of the accidents during a 6-year period in one of the American Steel plants were due to four causes; viz.: flying and falling objects 25.2 per cent.; falls of the worker 10.5 per cent., hot metal explosions and spills 8.9 per cent.; and the operation of cranes 8 per cent.

It was also found that there was a distinct excess in the number of accidents during night work amounting to 11.6 per cent. over the day rates. The data in reference to the influence of long hours on the accident rates were not sufficient to justify positive conclusions, but appear to indicate a tendency to greater frequency of accidents during the "long turns." A study based upon 26,839 accidents shows that for the forenoon of the 10-hour shift the greatest number of accidents occur in the fourth hour and for the night shift during the second and third hours of the work. This is contrary to the general expectation that accidents increase with increasing fatigue of the workers. Several reasons for this contrast are suggested in the report, viz., that during the early hours of the night turn much repair work is done necessitating overtime work of the day mechanics, hence also a larger working force during these earlier hours, and that because of the fatigue of these overtime workers and a tendency to hasten their work in order to get home or because of the exigencies of the case, the liability to accidents is increased. Another and quite reasonable explanation offered by the superintendents is that the men on the night shifts do not secure the same amount of restful sleep and that this less satisfactory physical condition tends to bring about a greater number of accidents during the period when "they are getting into the swing of the work."

A study of the seasonal distribution of accidents appears to justify the

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conclusion that both extremes of heat and cold have an influence, the heat condition being the more potent.

Prevention of Accidents.—A most gratifying reduction in the number of accidents peculiar to blast furnaces and rolling mills has been accomplished since 1905 by the application of safety devices. The rates for permanent injuries were reduced from 2.2 per 1000 in 1905 to 0.5 per 1000 in 1910; injuries to the eyes from 19.7 to 4.7; bruising injuries to the hands from 58.7 to 27.2; similar injuries to the feet from 46.4 to 22.8; bruises to other

FIG. 39—An emery wheel equipped with heavy steel guard. Guard on pulley and belt and permanent glass guard to prevent injury to the eyes. (Photo furnished to Würdemann by the U. S. Steel Corporation.)

parts of the body from 72.6 to 26.6; and burns from hot metal from 16.5 to 6.8 per 1000 workers. A chart on page 118 of the Government Report[8] shows a reduction in one of the large steel plants in the accident rate from 370 in 1900 to 109 in 1910.

Comparative Morbidity Rates.—We know that the morbidity rates are higher in the large steel and iron plants than in the manufacture of machinery and other mechanical departments of this industry. So, for example, the average number of men taken sick in the large steel plants at Solingen in 1905 was 476 per 1000; in the cutlery industry 404; and in the sword, bicycle industry, etc., only 302 per 1000 (Röpke[9]).

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Hayhurst[10] gives a synopsis of the chief occupational diseases in the iron and steel industry as follows:

1. Heat stroke (apoplexy), heat exhaustion, heat cramps, heat anæmia and heat diarrhea, premature senility in older employees.

2. Asthma, bronchitis, tuberculosis and hemorrhages while at work, due to dust, sand, gas and fume inhalations, grinding processes, etc.

3. Gas poisoning, producing chronic symptoms such as headaches, dizziness, vomiting, coated tongue, anæmia, palpitation, insomnia, general debility, mental dullness, and later depressive insanity.

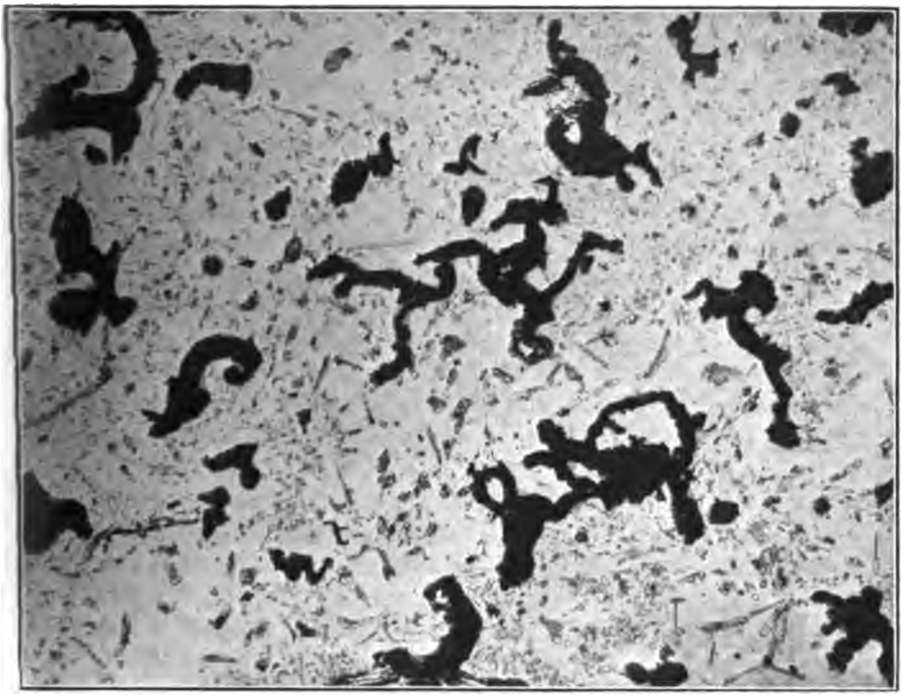

FIG. 40.—Steel dust to which steel workers are exposed. Particles magnified 40 diameters. (Photo furnished by Hanson).

4. Conjunctivitis, injected or bloodshot eyes, due to heat, sand and dust; cataracts among those exposed to white heated metals.

5. "Sunburn" of arms, hands, and face, due to exposure to heated metals. Small hemorrhages under the skin of the face; blistering, foot calluses, due to walking over hot iron plates, etc. Severe callusing of the hands.

6. Rheumatism and lumbago, due to great temperature variations.

7. Heart disease, perhaps evinced by sudden deaths while at work.

8. "Hammermans' paralysis" of arms, due to the use of heavy sledges, pneumatic tampers and other pneumatic tools, particularly with long strokes.

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9. "Strikers' arthritis" of wrist and elbows, due also to hammering or holding vibrating tools.

10. Boiler-maker's deafness. Ferrosilicon poisoning.

The U. S. census for 1910 gives the percentage of the chief causes of deaths for workers in this industry as follows: Tuberculosis 16.3; accidents 16.2; pneumonia 10.8; heart disease 10; Bright's disease 6.6.

The morbidity statistics in a very sanitary steel plant employing 5602 workers, presented by Hayhurst and based upon the records of a sick benefit association, but excluding accidents and venereal diseases, show that there were 1444 claims by workers sick for at least 5 days, or an annual percentage of 7.2. The claims for systemic diseases were as follows: Respiratory 391; digestive 285; communicable 218; musculo-osseous, mostly rheumatism and lumbago, 184; nervous, mostly neuralgia, sciatica, neurasthenia and neuritis, 79; skin 65; circulatory 63; urinary, mostly nephritis, 45; strain, mostly hemorrhoids, hernia, and heat prostration, 41; auto-intoxication and diabetes 27; chronic infections 18; special senses 17; lymphadenoids and neoplasm 11.

Average (Yearly) Morbidity in the Various Departments of an Iron and Steel Establishment During a Period of 3 Years—1911 to 1913 Inclusive

(The greatest variation of numbers employed did not exceed 15 per cent. in any one department)

Departments	Average number of employees	Average number sick	Average per cent. sick
(a) Heat exposed:			
Bessemer	393.3	33.0	8.39
Open hearth	145.3	7.0	4.81
Rail and shape mill	500.0	43.3	8.66
Blast furnaces	261.0	35.0	13.41
Foundry	163.3	12.7	7.77
Shelf mills	348.3	33.3	9.56
Pipe mill	1764.7	162.0	9.18
(b) Weather exposed:			
Police	35.0	2.7	7.62
Railroad (yards)	156.3	9.0	5.76
Section hands	110.0	10.0	9.09
Yard labor	468.0	28.7	6.13
Ore docks	90.3	8.3	9.19
Bricklayers	72.7	2.7	3.66
Building construction	106.3	9.7	9.07
(c) Indoors (mostly):			
Mechanical	472.3	47.7	10.09
Electrical	181.3	4.3	2.37
Miscellaneous	351.6	11.3	3.21
Total	5619.7	460.7	8.20

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Foundries.—The metal is reduced to a molten state either in a cupola, by combustion of coke, or in "open-hearth furnaces" heated with gas flames; in either case the operations involve exposure to intense heat and the inhalation of carbon monoxide. In tapping the molten metal there is considerable danger from explosions and flying sparks; in fact most of the serious burns occur in tapping either the slag or metal or during the transportation of the metal to the moulds and the process of pouring; other injuries are contracted in the moving of heavy castings, etc.

Accident Liability.—Among 16,480 foundry employees reported upon by the Bureau of Labor[11] there were 2198 accidents or 133.4 per 1000 workers. Of these, 8 proved fatal, 37 resulted in permanent disability and 2153 in temporary disability of 1 day or over; all of these rates are much lower than in the other departments of the large steel plants; injuries of the eyes were the most frequent accidents, next came bruises.

Hayhurst's Ohio Survey[12] supplies statistics for 1913 showing that the chief causes of death among foundry workers are pneumonia, heart disease, tuberculosis and violence. Sick benefits were distributed as follows: Accidents and burns 24.2 per cent.; rheumatism, sciatica and neuritis 21.8 per cent.; respiratory diseases 16.7 per cent.; gastro-intestinal diseases 10.4 per cent.; heart, kidney, liver, bladder 3.7 per cent.; tuberculosis 1.1 per cent., and the remainder for miscellaneous diseases.

Casters.—Apart from dust inhalation and the injurious factors already referred to, the casters are also exposed to organic vapors on account of the material of the cores, which are made by mixing sand with a "core binder" such as flour, sour beer, molasses, starch or some other sticky substance. The cores when completed are covered with a thin layer of pulverized coal or graphite which forms a vitreous layer while the metal is being poured. Contact of the hot metal with these moist moulds naturally causes combustion of the organic material with evolution of gases and steam.

The men engaged in casting generally wear smoked glasses to protect their eyes from the brilliancy of the light and sputtering of the metal.

Core and Mould Makers.—These employees are exposed to the inhalation of large quantities of dust, especially in sifting the sand for the cores and in dusting the completed moulds with powdered charcoal and graphite.

Female labor is often employed in the making of cores for small castings. The men engaged in breaking up the hot moulds naturally inhale large quantities of dust.

Ahrens[13] determined the presence of 28 mg. of dust per cubic meter in the air of the foundry room, and Hesse,[14] 71.7 mg. per cubic meter in the air of the cleaning and polishing room, which amount according to his calculation would subject each operative to the inhalation of 42 grams of dust per annum.

Apart from these dangers, foundries are notoriously dirty and gloomy workshops and the walls and windows are generally covered with dust and soot, shutting out light; very few are properly heated and in winter the mould

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and core makers who work in wet material often suffer from colds which are aggravated by drafts.

It is by no means infrequent to observe red hot cast-iron stoves giving off deadly coal gas or, what is even worse, open wood or coke fires, provided not so much for the comfort of the workers as for the drying out of the moulds and repaired fire-clay linings for the "pouring metal pots."

Fig. 41.—Machine shop workman using the movable burlap screen while chipping a piece of steel at bench vice. (Photo furnished Würdemann by the U. S. Steel Corporation.)

Cleaning and Milling of Castings.—In the removal of scales dilute sulphuric acid is used, the fumes of which are irritating. The subsequent processes of smoothing the castings by means of sand blasts, tumbling mills, emery wheels, etc., are all dust producing and have already been referred to on page 494.

In the department in which the castings are sand-blasted the air is fre-

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quently impregnated with flying sand which gets into the eyes, nose and mouth of the operatives, the effects of which few can resist for any length of time; most of the men have to quit within a year or two. Much of this unnecessary suffering is now prevented by thoughtful employers by providing in the center of the room a large flaring hood with upward suction draft, the operatives wearing helmets with fine wire inserts to protect the eyes, and cloths underneath the helmet for the protection of the nose and mouth. The latest pattern of helmets is provided with a tubing through which fresh air is supplied from the outside.

Unfortunately the vital statistics of foundry workers in this country are extremely defective. The Factory Inspector of Massachusetts[15] made a physical examination of 320 "moulders" in the Fall River District in 1910; of these, 103 or 32.2 per cent. were found to be in ill health, distributed as follows: Diseases of the lungs 12.8 per cent.; valvular diseases or dilatation of the heart 12.2 per cent.; diseases of the kidneys 7.2 per cent. According to Sommerfeld,[16] 41.7 per cent. of all the deaths among moulders in Germany are caused by consumption and 20.8 per cent. from other diseases of the lungs, such as asthma, bronchitis and emphysema. The consumption rate for casting cleaners was 35.7 per cent. and for other diseases of the lungs 24.3 per cent. Apart from the diseases referred to, rheumatism, digestive disorders, affections of the skin, eyes and ears are also quite common.

Preventive Measures.—There can be no question that the factors referred to render employment in this industry more or less injurious to health. It should be remembered, however, that bad working conditions are responsible for much of the ill health. That present conditions are in need of and susceptible of betterment is strikingly evinced by the following comments of medical inspectors.[17]

"In one of the establishments the dust from the polishing and buffing process in the absence of hoods and exhaust ventilation was very great and in the tumbling room was so thick that objects a few feet distant cannot clearly be made out. Many men refuse to work in this establishment on account of the excessive heat and general discomfort."

In another establishment the general surroundings were extremely favorable, the room in which the castings were cleaned and tumbled was large and airy; in the polishing room the emery wheels were provided with hoods and exhaust ventilation, but the men ignorant of the dangers habitually removed the hoods, maintaining that the hoods interfere with their work and that "iron dust is strengthening as it enriches the blood."

It is gratifying to note that thoughtful foundry owners[18] appreciate that health and efficiency can be promoted by providing suitable workrooms, properly heated and equipped with mechanical devices for the removal of dust, gases and fumes; they also furnish pure drinking water and good washing and bathing facilities. All others should be required to do like-

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wise, and when thus provided the workmen should be obliged to avail themselves of these safeguards.

Blacksmiths, Forgemen, Boilermakers, Riveters, Structural Iron Workers, Etc.—Space will not permit a consideration of all the occupations connected with the iron industry, and hence only a few of the more hazardous trades can be considered.

Blacksmiths and forgemen, apart from hard work and exposure to intense heat and abrupt changes in temperature, are also exposed to the inhalation of coal dust, smoke, fuel gases and the fumes evolved during the tempering and case hardening with lead, potassium cyanide and oil. There is also danger from flying scales which not infrequently affect the eyes.

Wounds, contusions, burns, fractures and hernia are not infrequent. They are also liable to conjunctivitis and skin affections caused by exposure to radiant heat and deposits of dust. Lumbago and acute rheumatism are frequently met with. According to Sommerfeld[19] 66.6 per cent. of all the deaths in this class of workmen are caused by consumption and 8.4 per cent. by other diseases of the lungs.

The tuberculosis death rate, while very high in Germany, is not excessive in this country. According to U. S. census, 11.4 per cent. of blacksmiths died of pulmonary consumption, 8 per cent. of pneumonia, 13.8 per cent. of heart disease, 10 per cent. of apoplexy and 9.7 per cent. of Bright's disease.

Hypertrophy of the heart, diseases of the aortic valves and of the kidneys are not uncommon. When most of the heavy work was done by manual labor instead of machinery, rupture of muscular fibers of the upper arm and shoulder and cases of so-called "hammer's paralysis" caused by overfatigue of the muscles of the arm were frequently met with. Even now occasional cases may be observed in forgemen, and likewise instances of "strikers' arthritis" which is a painful inflammation of the wrist- and elbow-joints.

The men engaged in handling sledge hammers, chisels, files and other tools are also liable to develop disagreeable callosities and thickening of the fascia with consequent contraction of the fingers toward the palm of the hand. Faulty positions, stooping over anvils, work benches, etc., are a frequent cause of lateral spinal curvature and of genu valgum ("knock-knee") in youthful workers and should be prevented by teaching apprentices at the outset correct positions.

The work of boiler makers and structural iron workers is also laborious and attended with more or less danger from accidents. As a result of hard work and exposure to abrupt changes in temperature they are liable to the development of rheumatic, neuralgic and catarrhal affections and diseases of the respiratory and circulatory organs. In riveters using a hand hammer spasm and paralysis of the muscles of the hands are liable to occur, while the constant jarring of the automatic hammer has been known to produce occupational neuroses. Progressive impariment of hearing is by no means confined to boiler makers and riveters since Gottstein and Kayser[20] found that fully

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61 per cent. of the blacksmiths and locksmiths examined suffered from partial deafness.

The Cutlery and Tool Industry.—This extensive industry involves a number of processes, such as iron and brass founding, forging, blacksmithing, welding, tempering, grinding, polishing, buffing, pickling, acid dipping, enameling, etching, electroplating, tinning, wood working, painting, varnishing, etc. The health hazards are peculiar to the different processes named.

FIG. 42.—Grinding iron castings. Employee is wearing goggles to prevent injury to his eyes. (Photo taken by Hanson for the Mass. State Board of Health.)

Steel Grinding and Polishing.—In the grinding and polishing processes of the metal industry in general, and in the manufacture of cutlery, tools and steel implements in particular, immense quantities of dust are evolved, not only from the metallic surfaces but also from the numerous grindstones, revolving wire brushs, emery and corundum wheels and other buffing material. Professor Roth[21] estimates that a grindstone 2 meters in diameter and 10 cm. in width after 4 months' constant use is reduced fully one-half, and the various metal implements lose from 20 to 30 per cent. of their original

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weight during the grinding process. This affords an approximate idea of the amount of dust production, which is not always wholly avoidable, even by the employment of hoods and exhaust ventilation or by the wet process, especially during the "racing" of the grindstones.

It is well known that the inhalation of the hard sharp and angular particles of steel, sand, an emery dust produce a very intensive irritation of the mucous membranes of the nose and air passages, causing not only catarrhal conditions, but also more serious chronic inflammations of the respiratory organs, such as bronchitis, peri-bronchitis, fibroid pneumonia, also spoken of by the workmen as "grinders' asthma" and "grinders' rot." The chronic inflammatory conditions thus produced, together with exposure to dampness and other injurious factors, naturally favor infection with the tubercle bacillus.

Hirt gives the percentage of consumption in the total number of sick among different classes of metal workers as follows: Needle polishers 69.6 per cent.; file cutters who are also exposed to lead poisoning 62.2 per cent.; grinders 40 per cent.; and nail cutters 12 per cent.

Beyer[22] found that of 196 needle polishers at Remsheid only 24 were over 40 years of age. The reason why this occupation is especially dangerous is because the "wet process" cannot be employed for small objects, which moreover have to be brought more closely to the eyes, and thus the chances for the inhalation of dust is increased.

Hoffman[23] reports that of 128 deaths among grinders 63 or 49.2 per cent. were due to industrial tuberculosis as compared with 14.8 per cent. in the total male population over 15 years of age in the U. S.; 15 deaths were caused by pneumonia; 5 by asthma and bronchitis, and 2 by other diseases of the respiratory organs, a total of 85 deaths or 66.4 per cent. from diseases of the lungs.

The death returns for 12 years of the city of Northampton, Mass.,[24] one of the centers of the cutlery and tool industry, show that among "grinders," "polishers," and "cutlers" diseases of the lungs were responsible for 72.73 per cent. of the mortality, inclusive of 54.5 per cent. of deaths from tuberculosis.

During 1850–1874, according to Oldendorff quoted by Röpke,[25] the mortality among the grinders and polishers over 20 years of age of Solingen, the seat of the cutlery industry in Germany, was 25 per 1000, as compared with 12.6 in the general male population. From 1885–1895 the relative figures were 20.63 per 1000 among the grinders and polishers and 13.6 in the general male population over 20 years of age.

According to Dr. Scurfield, Medical Officer of Health of Sheffield in England, the mortality rate of the steel grinders over 18 years of age between 1901–1909 was 30.4 per 1000 and in the male population over 20 years of age only 16.4 per 1000; the tuberculosis death rate was 15.1 and from other diseases of the lungs 5.4. Most commendable progress has been made

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at Solingen, where according to Röpke the mortality has been reduced from 20.63 in 1885 to 9.3 per 1000 in 1910 and the mortality from diseases of the respiratory organs from 12.82 in 1885 to 6.72 per 1000.

In addition to the dangers referred to, the "grinders" not infrequently sustain injuries from bursting grinding stones and polishing wheels while in motion, for which adequate protection has been found in the case of the smaller wheels by a strong hood which incloses the wheel except at the point where the grinding is done, while for the larger wheels a special form of wheel with a safety collar is used.

The "wet process" is not by any means free from danger. While dust production is reduced to a minimum, the constant throwing off of water saturates the clothing, floors and air and predisposes to "colds" and rheumatic conditions, especially when the operatives are careless about their clothing.

Some of the grinders also assume a faulty position, crouching over their work, which interferes with the free expansion of the lungs.

Preventive Measures.—Apart from securing general sanitary working conditions, every effort must be made to diminish the amount of dust production to a minimum by substitution of the "wet process" as far as practicable. In Solingen the "dry grinders" furnish 13.7 per cent. of the lung diseases, the "wet grinders" 11.3 per cent. In all "dry grinding processes" forced ventilation by means of hoods connected with a system of exhaust fans or blowers to carry away the dust from the operator should be installed and their faithful use insisted upon.

Unfortunately, in this country at least, a large proportion of grinders, prefering freedom of movement, remove the hoods and thus expose themselves unnecessarily to this especially dangerous form of dust. This state of affairs can be overcome only by general education and legislation. Scrupulous cleanliness of the person, clothing, tools and work benches should be observed. The employees should be taught from the outset proper positions and the habit of breathing through the nose. Abnormal conditions such as adenoids, enlarged tonsils, etc., necessitating mouth breathing, should be removed.

The results of ameliorating efforts are strikingly shown not only by the decreased mortality rates at Solingen, but also by the fact that morbidity and loss of work among the insured grinders has been reduced from 38.5 per cent. in 1886–1898 to 22.6 per cent. in 1902–1904. Grindstones should be inspected in order to detect flaws, properly mounted and secured by means of plates and bolts and the safety devices already referred to. For general and individual hygienic measures see also pages 428, 429.

Tempering.—The process of tempering or hardening of metals is carried on very extensively in the metal industry, especially in the manufacture of cutlery and tools, springs, files, foundry and machine-shop products, automobile and bicycle parts, sewing machines, stamps and stencils, cash registers,

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electric apparatus, etc. This process is not dangerous to health when it involves simply heating and cooling, or heating and dipping into water or brine. On the other hand, the dipping into hot bed of lead, oil, or potassium cyanide, or the application of potassium cyanide, and subsequent heating in an oven, involve serious risks unless done with special precautions and adequate exhaust ventilation. A patented preparation advertised recently as a harmless substitute for "case hardening" is said to yield the poisonous cyanide radical when melted for use.

File Cutting.—It is to be regretted that machine work has not wholly supplanted the dangerous process of hand file cutting. It is claimed that the best files are cut by hand, and as long as there is a demand, manufacturers will continue to supply it regardless of the health of the workers. This occupation appears to be the most dangerous of all dusty occupations; the comparative mortality rate in England is given by Dr. Tatham[26] as 1810, practically three times higher than that of agriculturists. This excessive mortality is due to a combination of causes, viz.: 1. The laborious character of the work incident to the use of the steel hammer, which weighs from 8 to 20 lb., and during an average day's work amounts to lifting over 150,000 lb. weight. 2. The work is performed in a stooping position, which interferes with normal respiration. 3. Both the smoothing process of the blank files and the process of cutting the lines by means of a chisel and hammer involves the inhalation of steel dust. 4. While the file is being cut it is usually held upon a leaden bed to prevent slipping and a sharp recoil and also to protect the already cut surface from injury. During this process particles of lead may be inhaled or swallowed with the dust, or they may be taken up by the fingers in brushing off the file, or in handling the bed, and carried to the mouth in licking the fingers and left thumb to secure a firmer grip of the chisel. Another source of lead poisoning is that the files are hardened by passing them through kettles of molten lead. From 3 to 6 per cent. of lead have been revealed in the dust of file-hardening establishments.

The Ohio Survey of 1914 dislcosed cases of lead poisoning, occupation neurosis from hammering, rheumatism, stiff wrists, diseased gums and teeth, and inflammation of the margin of the eyelids.

According to Sprenger, cited by Röpke, 5.24 per cent. of the file cutters in Berlin using the lead bed suffer annually from plumbism.

Preventive Measures.—Efficient exhaust ventilation and adjustable seats and work benches are indicated. All sorts of substitutes for the lead beds, such as pads of paper, wood, clay, sand, gutta percha, etc., have been tried and finally block tin has come into use, which Röpke declares to be free from objections both from the hygienic as well as technical point of view.

Gunsmiths, Firearms, Weapons.—The mortality figure of gunsmiths according to Tatham is 1228, or 100 in excess of that of metal workers in the aggregate; the mortality from phthisis is 324 as compared with 206 among other metal workers, and 185 for all other occupied males; the death rate

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from other diseases of the lungs and the heart is also excessive. Tatham regards most of the processes in which they are engaged as injurious, especially the exposure to the inhalation of metallic and mineral dust during the filing and polishing of the metal. The occupation is often carried on in insanitary shops, and the employees in England, otherwise high-grade men, appear to suffer from the alcohol habit.

The men employed in the manufacture of *firearms*, in the metal department, apart from the inhalation of metallic dust, not infrequently are exposed to nitrous gases evolved in the etching process, to vapors of antimony compounds in the burnishing of rifle barrels, and in the steel bluing room to the fumes of charcoal, giving off carbon monoxide. In the manufacture of gun stocks, large quantities of fine black walnut dust is evolved.

In the manufacture of swords, sabers, bayonets, etc., apart from the dangers incident to steel grinding and polishing, the men engaged in fire gilding are exposed to the risk of mercurial poisoning.

Agricultural Implements.—Automobiles, motorcycles, bicycles, sewing machines, cash registers. In the manufacture of these goods various processes are involved; some are characteristic of the metal industry, such as brass and iron founding, blacksmithing and forging, welding, bronzing, tempering, grinding, polishing, buffing and dipping, electroplating, etc. There may also be more or less woodworking and painting, with all of the hazards attending these operations.

Iron Sanitary Ware Factories.—The dangers to the workers employed in the manufacture of so-called porcelain or vitreous enameled iron ware, such as bath tubs, sinks, basins, gas stoves, hollow ware, iron plates for sign boards, etc., are the elements of overfatigue, excessive heat, and exposure to dust containing poisonous lead compounds. This industry, as conducted at present in this country, must be regarded as extremely dangerous. In Germany the enamel employed is leadless, while in this country it contains from 2½ to 25 per cent. of lead. (See also page 111.)

The Wire Industry.—When the ingots are ready in the "open-hearth department" of wire mills they are rolled into long rods and placed upon large spools or reels, from which the rod is carried through the drawing block and thence to another revolving drum drawing the rod through the block. This process is repeated until the desired size of the wire is reached. The workmen are protected to a certain extent from the heat by metallic screens over which water trickles, but cases of heat exhaustion are not uncommon, especially in summer. The process is quite dusty from the lime which is put on the wire to prevent rusting. The occupation also involves considerable liability to accidents as a result of the fingers getting caught between the wire and the drum in drawing from the block. It not infrequently happens that in passing to the block the moving wire becomes tangled and the operator is caught and drawn toward the block. Unless the machine can be stopped the workman is likely to be seriously injured, the loss of a

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hand or a foot being often recorded. To guard against this possibility an automatic stop has recently been introduced and proved to be an effective safeguard.[29]

After the wire is hardened by being run through crude oil it is passed through kettles of molten lead inside the tempering furnaces, and the men who feed and tend the furnaces not infrequently suffer from chronic lead poisoning, a danger wholly avoidable by efficient mechanical ventilation, which would carry these fumes to the outer air.

Other sources of danger in the manufacture of wire are the employment of sulphuric acid for annealing and pickling purposes, the coppering process by the employment of sulphate of copper. The dangers from carbon disulphide and lead incident to the manufacture of insulated wire and cable should also be mentioned.

In the process of *galvanizing wire* it is first passed through diluted commercial sulphuric acid, next through a weak solution of hydrochloric acid and finally through molten zinc (see page 502).

Preventive Measures.—It is needless to insist that adequate provisions should be made to carry off the acid and metal fumes rapidly into the outer air. The workmen should be instructed to avoid the fumes as much as possible and to annoint the lips and nose within and without several times a day with lanolin.

Wire Cloth, Netting and Fencing Material.—In the manufacture of these materials the wire is first rolled on spools and drums to form the warp and is then woven by means of heavy looms. There is no special danger in this industry unless the japanning is done without adequate exhaust ventilation. The latter process is usually carried on in a suitable tower, the cloth passing from the rolls through a vat of japan, and as it emerges is subjected to the action of a blower, then passing upward through several stories, where it is dried by steam heat, and finally descending on another side to be wound again into rolls.[30]

Tin Industry, Tin Mining.—Dr. Ogle, the Registrar General of Great Britain, was perhaps the first to point out that the consumption rate among the Cornish *tin miners* was far in excess of any other class of miners; other diseases of the lungs are also more common. Consumption is the cause of 29.9 per cent. of all the deaths, bronchitis 13.5 per cent.; pneumonia and other diseases of the lungs contribute 12.5 per cent. of the mortality. The death rate in 1904 in miners between 25–45 years was eight to ten times higher than among coal and iron-ore miners of the same age. The increased mortality in recent years is attributed by Oliver to the increasing employment of rock drills, and to the fact that a good many men have worked previously in the quartz mines of the Transvaal.

Since tin is not regarded as a toxic metal like lead, the undue prevalence of diseases of the respiratory organs has generally been attributed to the inhalation of the hard, sharp and angular particles of dust.

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Cases of tin poisoning have been reported, but it has never been satisfactorily shown that tin was the real cause of the intoxication. On account of the widespread use of tin, especially in connection with the canning industry, Dr. Salant[31] of the U. S. Dept. of Agriculture has recently undertaken an investigation as to the behavior of tin in the body. Animal experimentation has shown that absorption of tin from the gastro-intestinal canal is not only possible, but that repeated ingestion of the salts of tin produces changes in the alimentary mucosa which favor the absorption of tin. Animals which had received daily doses of sodium stannous tartrate showed no tin in the urine until after the first week. When tin was introduced into the circulation, the gastro-intestinal tract appeared to be the chief organ for its elimination. The tin disappears from the blood in 2 or 3 hours after an intravenous injection of its salts, and can be found in the liver, brain and muscles, showing conclusively that when it actually reaches the circulation its distribution becomes very general. It is hoped that these experiments will be continued, in order to clear up the toxicologic problem of tin.

Tin containing about 10 per cent. of lead was at one time quite extensively used for the manufacture of trophy cups, coffee and tea pots, plates, spoons, art goods, toys, etc. The use of pewter, German silver and Britania metal has to a considerable extent displaced the manufacture of the former tin ware. The dangers in the industry are incident to the polishing processes and also from lead poisoning, which is proportioned to the amount of lead used.

Tin plating on iron or steel sheets has become a large and still growing industry, in which more or less female and youthful laborers find employment. The material is used for roofing purposes, the manufacture of tin cans, boxes, hollow ware, patent bottle caps, etc. Culinary and domestic utensils like saucepans and iron kettles are also tinned. The general process of tin plating is similar to the galvanizing with zinc, except that the metal requires more careful cleaning and smoothing of its surfaces. The pickling with a weak solution of sulphuric acid is followed by dipping into a solution of weak hydrochloric acid and zinc chloride. The metal is then dipped into a bath of molten tin, but very often the percentage of lead exceeds that of the tin, and hence the operatives in addition to the exposure to acid and zinc fumes incur the risk of lead poisoning.

Oliver[32] points out that the cheaper ware is frequently dipped in a composition of 70 per cent. of lead and 30 per cent. of tin, thus exposing not only the workers but also the purchaser of such utensils to the danger of lead poisoning. A number of cases of lead poisoning have likewise been reported in consumers of canned foods, especially of acid fruit, and even carpenters and shoemakers have contracted lead poisoning from holding so-called tin-plated nails or tacks in their mouths. It is a satisfaction to learn from Dr. Alice Hamilton[33] that while the manufacture of cheap kitchen ware is a bad lead trade in England, the ware in the United States is tinned without lead.

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The washing and dipping is extremely wet and disagreeable work, and the subsequent polishing involves inhalation of more or less dust of a mixed character. The acid dips, unless the hands are protected with rubber gloves or the articles are handled with wooden tongs, are liable to produce obstinate eczema, suppurative pustules and even symptoms of blood poisoning. Neisser[34] reports a number of such instances requiring from 6–8 weeks' treatment.

Tinners, Tinkers, Etc.—The work of roofers, tinners and tinkers involves the use of solder. Apart from the danger of lead poisoning, there is exposure either to carbon monoxide gas from open charcoal fire or the products of combustion from the gasoline torch, also to the irritating fumes of hydrochloric acid and possibly arseniuretted hydrogen, if the acid happens to be derived from arsenical pyrites. The latter risk although slight may also be encountered in zinc, tin and lead-plating works.

The handling of tin plates, with their sharp cutting edges, is very apt to cause injuries of the hands unless they are properly protected. Roofers are often exposed to the elements and as a result many contract colds, catarrhal and rheumatic affections, and inflammation of the middle ear. Burns from the soldering iron, etc., are not infrequent. Acute and chronic conjunctivitis from dust, foreign bodies, and exposure to acid fumes is also common. The morbidity hazard of the Leipsic tin-ware factory workers is 827 days of sickness per 100 members per annum and the mortality hazard 0.79 per cent.

Since many articles of tin ware are also painted and lacquered, the element of danger from toxic color pigments and varnishes must likewise be considered. In recent years the process of mixing copal varnish is done by means of compressed air, with efficient exhaust ventilation, so that the workers are no longer exposed to the inhalation of this dust.[34]

Tin foil, which is so frequently used for bottle tops and wrappers for cheese, tobacco, snuff, cigarettes, etc., should be spoken of as lead foil as it contains generally from 85 to 95 per cent. of lead. The manufacture of lead foil should be discouraged as it exposes the workers to lead poisoning and is not wholly free from danger to the consumer.

Vanadium.—This rare metal, a silvery crystalline substance, is obtained from vanadinite and other minerals. The pentoxide is used in photography as a developing agent; the chloride and trioxide are used as a mordant in the textile printing industry. The trioxide is also used in the manufacture of malleable, ductile steel, such as is used for making the chassis of automobiles. According to Dr. Dutton,[35] persons employed in reduction plants or where the various kinds of vanadium, especially the trioxide, are produced are liable to suffer from chronic poisoning. Among the early symptoms he mentions a peculiar cachexia not unlike chlorosis. Irritation of the nose, eyes and throat and a dry paroxysmal cough sufficiently intense to cause pulmonary hemorrhage are frequent symptoms. There is also pro-

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gressive emaciation, loss of appetite, nausea, diarrhea, followed by obstinate constipation. Albumin casts and blood corpuscles are often present in the urine. The metal is eliminated by the kidneys and has also been detected in the feces, saliva and sputum. Continued exposure to the poison may produce tremors of the extremities, neuroretinitis, amaurosis, hysteria and melancholia. In fatal cases Dr. Dutton[35] found the lungs highly congested with marked destruction of the alveolar epithelium; the kidneys were congested with evidence at times of an acute hemorrhagic nephritis; there was also more or less evidence of gastro-enteritis. While O. T. Cruckshank has produced similar lesions in animal experimentation with vanadium, Lees[36] failed to find that there is such a disease as vanadiumism. Dutton, however, is a man of large experience and his observations should carry great weight. He holds that a fatal termination frequently ensues as a result of continued exposure to the poison, but that the prognosis is good in the absence of grave renal, lung, blood and nervous involvement.

Treatment.—In the way of treatment he suggests washing out of the stomach and intestinal evacuation by saline laxatives. For the relief of cough, terpin hydr. ¼ gr., heroin ⅛ gr., creosote ½ ℳ every 2 hours and counter-irritation over chest, also inhalation of steam vapors. Turkish, Russian or cabinet baths have proved useful to aid in the elimination of the poison, while in the less acute cases the administration of iron, calisaya, strychnine, cod-liver oil, and outdoor exercise are of service for the relief of the anæmic, nervous and debilitated condition.

Preventive Measures.—Perfect ventilation and exhaust flues for the removal of dust and fumes, and the use of respirators, are essential. The nasal and oral cavities should be cleansed before meals and upon cessation of work by means of a spray with Dobell solution, followed by a mentholated oil spray. Change of work upon appearance of suspicious symptoms is indicated.

Robinson and Wilson[37] examined 2860 workers in *Foundry and Machine Shop Products* in Cincinnati, and diagnosed 27 cases of tuberculosis (0.94 per cent.). About 44 others were below normal health for this occupation. The hazards, such as all kinds of dust, cold, dampness, fatigue, and noise vary in degree and depend not only upon the particular process, but also upon the safeguards in force and the sanitary and hygienic conditions maintained by the employer. This survey revealed gratifying progress in the sanitary and hygienic conditions, and also that there is need for further improvement.

Among 565 persons employed in the *Tin and Sheet Metal Industry*, 12 cases of tuberculosis were found. Many of the men in the galvanizing department were found below par and in one of the three establishments visited no exhaust was provided to carry off the fumes, and certain parts of the factory were cold and damp. Spitting on the floors was found in two of the places visited. Piecework and speeding up was also in evidence.

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CHAPTER V

THE PRINTING AND PUBLISHING INDUSTRY

BY GEORGE M. KOBER, M. D., Washington, D. C.

Type Metal. Type Founding. Typesetting. Monotyping and Linotyping. Stereotyping. Electrotyping. Lead Melting. Binding-room processes. Chromo-Lithography. Engraving. Metal Etching. Photography. Preventive Measures.

Printers.—During the year 1909, the printing and publishing industry in this country afforded employment to 388,446 wage earners. Of these, 77.7 per cent. were males and 22.3 per cent. females. The average number of wage earners under 16 years of age was 6333, of which about one-sixteenth were girls. These figures cover the industry as a whole, which includes music-printing, book binding and blank-book making, engraving on steel and copper plates and printing from such plates, and lithographing.

The morbidity and mortality of printers is everywhere very high. The statistics of German printers based on a membership of 54,000 from 1891–1904 gave a sickness rate of 461 per 1000 as compared with 375 in the occupied male population.[1] According to Schuler, of 1000 Swiss typesetters and founders 304.7 are anually taken sick, and of printers 250. Diseases of the digestive organs predominate (78 per 1000). Diseases of the respiratory system come next (75 per 1000).

The death rate per 1000 employees engaged as compositors, printers and pressmen in the United States was 12.1. Among 27 occupations tabulated by the Census Bureau these workers occupy the third rank in the number of deaths from tuberculosis, and in 38 occupations tabulated by Professor Sommerfeld the German printers occupy the fifth rank. Albrecht[2] reports that the statistics of the Berlin Sick Benefit Insurance Companies, covering a period of 33 years, show that 48.13 per cent. of all the deaths among printers prior to 1887 were caused by consumption. The statistics collected since that time by Sommerfeld and Silberstein[3] are more favorable, since only about one-third of the deaths among the Berlin printers in 1903–1905 were caused by consumption. This gratifying reduction is doubtless the result of improved sanitation in the workrooms as required by regulations of the Federal Council of the German Empire which went into effect July 31, 1897. At all events, in a total membership of 44,236 among the printers of Germany in 1905 only 134 or 3.03 per 1000 died from pulmonary diseases (tuberculosis not being separated in the tabular presentation). The corresponding death rate among compositors in New York City was 7.17; in the State of New York 4.04; total New York State 6.34; Chicago 5.04; Philadelphia 4.70, total

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United States 5.02 and London, England 5.50. See interesting study of the health of printers by George A Stevens.[4]

The undue prevalence of consumption among printers may be due in part to the fact that many weaklings engage in this occupation, but the work itself is doubtless an important predisposing factor, as it is is often carried on under most unfavorable environments and in an atmosphere rendered impure by the products of respiration, combustion and presence of type metal, plumbago dust and benzine vapors evolved for cleaning purposes.

Type metal is an alloy containing about 75 per cent. lead, 23 per cent. of antimony and about 2 per cent. of tin, copper or zinc. During the wear of the type more or less dust is given off which generally collects at the bottom of the type cases. One gram of this dust upon analyses revealed the presence of 57.5 mg. of lead, 186.8 mg. of antimony and traces of arsenic.[5]

According to Fromm[6] the dust collected from type cases contained 38.77 per cent. of lead, the dust deposited in compositors rooms contained 1.95 per cent. of lead and the floating dust in such establishments revealed the presence of 0.54 to 1.55 per cent. of lead. Other investigators like Panwitz[7] detected only traces of lead in the atmospheric dust of compositors' rooms.

In addition to this, more or less plumbago is used in electrotyping which is not always sufficiently removed before the type is redistributed. It will be readily understood that the inhalation or ingestion of this metalliferous dust not only predisposes to diseases of the respiratory organs but also causes lead poisoning in various degrees, resulting at least in such a general deterioration of the health of the workers as to render the system susceptible to the invasion and effects of tubercle bacilli. At all events, Professor Hahn[8] points out that the workers both in Berlin and Vienna between 1901 and 1907, who are most exposed to plumbism, viz., the printers and type founders, also show the highest mortality from tuberculosis. Cases of pronounced lead poisoning are not very common. According to Sir Thomas Oliver[9] only 200 cases were reported in printers in Great Britain between 1900 and 1909. Conditions are, however, worse in Germany as in Leipzig in 1 year alone 132 typists, 25 type founders and 9 music typists suffered from lead poisoning. In Vienna, between 1891 and 1900, 1308 cases were reported with 41,838 days of sickness and 8 deaths, exclusive of about 400 cases of a milder type which did not incapacitate the individuals for work. Females and youthful employees are especially susceptible to lead poisoning. The processes in which the workers are especially exposed to injurious factors are the following:

Type Founding.—It was held for a long time that the old process of casting type involved exposure to lead and arsenical fumes, as the antimony frequently contains arsenic. This, however, according to Sternberg,[10] is not the case as the melting point of lead is 326°C. and the vapor point is over 1000°C.; the latter point is never reached in this process.

Schuler, cited by Silberstein,[11] has pointed out that the so-called lead

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fumes complained of by type founders are acrolein vapors, due to the presence of wax, fat or oils used in fluxing the metals (see Acrolein). The polishing of the type by means of files and machines is usually done by female labor and youthful employees and involves exposure to lead dust, and the sorting and packing, also largely done by female labor, involves at least the risk incident to the constant handling of a lead-containing metal.

Typesetting.—The compositor is chiefly exposed to the inhalation and ingestion of the dust from the type cases; the absorption by the skin is probably insignificant as only the apex of the thumb and index-finger are brought into contact with the metal in setting up or distributing of the type; the skin of these fingers soon becomes hardened, rendering the chances of absorption quite remote. On the other hand, it should be remembered that some compositors, especially youthful members, are in the habit of moistening their fingers in order to get a better hold of the type, and of holding the type between their teeth while correcting proof. This, together with smoking and an equally pernicious habit of eating fruit and lunches with unwashed hands, is a frequent mode for the ingestion of lead. Prof. Van Eyk, cited by De Vooys and quoted by Oliver,[12] requested a number of printers, after several hours' work with type, to wash their hands in a weak solution of hydrochloric acid and found on a average from 6 to 15 mg. of lead in each wash basin.

The danger from type case dust could be materially reduced by proper construction of the cases. Strasser, nearly 30 years ago, suggested a type case with a perforated tin bottom, placed within another case; the dust would naturally drop from the upper to the lower case, and if the latter was provided with a concave and rounded bottom its collection and removal would be materially facilitated, especially since the introduction of vacuum cleaners. In spite of this practical suggestion, the writer has never seen such cases in use, and even in the Government Printing Office in Washington, an otherwise high-grade institution, the old style of cases are still in use, but are now cleaned by the vacuum system instead of by bellows as formerly.

Monotyping and Linotyping.—Both of these processes, the former casting single letters and the latter an entire line, have superseded in all large printing establishments the old methods of type founding and typesetting. The work is nearly all done automatically by machines and hence the danger from dust, or handling of the type, has been greatly reduced. For reasons already given there is no danger from lead or arsenical fumes from the melting pots. The operatives, however, are exposed to irritating acrolein vapors from materials used in fluxing the metal, such as wax. In monotype machines which operate directly over the metal pots, oil from the machine is liable to drip into the molten metal and aggravates the offensive acrolein vapors. All of this should be obviated by suitable hoods and exhaust flues connected with each pot; this method, however, must be regulated so as not to produce an excessive draught and thereby overheat the metal.

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Stereotyping.—In the process of stereotyping the type forms are placed in steam or gas, or preferably electric heated presses, for the purpose of making a negative impression upon paper specially prepared for the purpose. The impression or matrix is placed in suitable moulds, into which the molten metal is either poured by a ladle or pumped automatically, until the desired thickness of the slab has been attained. This process, as well as the preliminary act of melting, necessarily involves handling of the lead and exposure to its toxic effects.

The formed plates are subsequently trimmed. Dr. Manning of the Government Printing Office believes that the finishers of the plates, who handle only the smooth, hard, bright slabs run the least risk of lead poisoning.

Electrotyping.—In this process an impression of the set-up type is made upon a layer of wax, both the form and the wax having previously been covered with plumbago to prevent adhesion of the surfaces. The wax matrix is then placed in a closed box and more plumbago is blown on it by means of compressed air, which is an extremely dusty process. Recently modern establishments have adopted a wet method which consists in the application of a liquid graphite-paste by means of a hand brush, or by having the matrix move automatically upon an incline, back and forth through the graphite mixture. Dr. Manning informs me, that by this method the dust production has been reduced fully 90 per cent. in the Government Printing shop. Iron filings, followed by a solution of sulphate of copper, are next applied to the wax impression to form a plate, which is then immersed in the electrotyping solution to secure the desired thickness of copper deposit. When this is attained the copper plate is removed from the wax and thoroughly cleaned. It is then placed face downward in a tray and backed by pouring electrotype metal lead on it; all of which necessarily involves considerable handling of lead and exposure to irritating fumes.

Lead Melting.—The men engaged in melting the new metal and old slugs are exposed to the risk of plumbism from handling the metal and breathing its toxic fumes if the temperature should reach the vaporizing point. The temperature of the rooms in which this process and type founding is carried on is also frequently above 86°.

In the Government Printing Office the melting is done in special rooms after the usual working hours and the men are shifted to other work at the expiration of 1 week. The melting pots are placed between open windows so as to insure copious ventilation, aided by revolving fans. It is said that the hoods interfere with the stirring of the metal.

Summary.—The chief occupational diseases in the printing industry are tuberculosis, other diseases of the respiratory system, and plumbism. Diseases of the digestive organs are also quite common, and compositors and proofreaders not infrequently develop neurasthenia. Compositors, and all others obliged to maintain a standing position, are liable to develop flat-foot, varicose veins and ulcers. There is also evidence to indicate that the

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married women employed in this industry, probably as a result of lead intoxication, miscarry more frequently than women engaged in other occupations. According to Silberstein[13] the percentage in Berlin in 1905 was 2.4 and 1.7 respectively. According to the same author inflammatory diseases of the skin are also more common in the printing industry, viz., 20 per 1000 against an average of 13 in other occupations. Eczema of the hands and forearms in the workmen who handle the type-forms is evidently caused by the irritation of certain agents such as turpentine, petroleum, benzine, lye, etc., employed for the removal of printer's ink. Quite recently Zellner and Wolff[14] have called attention to the undue prevalence of these skin affections, and attribute them to trade substitutes for the oil of turpentine, which was formerly almost exclusively employed. Among the substitutes were found inferior benzine, lye, poorly refined petroleum, and impure turpentine, called "pine oil." Of 37 samples of "pine oil" and other cleaning materials examined 32, or 87 per cent., contained impure benzine or lye, and the turpentine used was also found to be adulterated with impure benzine. It is well known that even pure oil of turpentine and refined petroleum may prove irritants to sensitive skins, but they are at present the least objectional agents employed and every effort should be made to suppress the use of lye and irritant adulterants or substitutes. The Government Printing Office provides lye and the men are enjoined to immerse their hands subsequently in a solution of vinegar, while those working in acid dips are expected to neutralize the acid by immersion into a weak ammonia solution, but this method has not wholly prevented the occurrence of eczema.

Binding Room Processes.—The work is generally carried on in unfavorable surroundings, as regards light and air. Dust, heat and exposure to wood alcohol, from the shellac employed, constitute the chief injurious factors. Persons employed in folding heavy paper with their hands are liable to develop cysts of the wrist tendons in consequence of constant local pressure.

Preventive Measures.—In view of the foregoing it is evident that the utmost precaution should be taken to reduce the dangers in printing establishments to a minimum. All processes involving the handling of molten metal should be carried on in the upper stories or in separate buildings, to facilitate the removal of injurious fumes by means of hoods and exhaust ventilation.

Cleaning of electrotype plates for the removal of caked plumbago should be done by means of steam in a "boiling chamber" and the plates lifted in and out by hooks. This method, in vogue in the Government Printing Office, has reduced dust production to a minimum and also secured a thorough removal of the graphite. Perfect cleanliness of the establishment aided by vacuum cleaners should be observed.

Light, air space, general ventilation, temperature, humidity, spittoons

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and facilities for personal cleanliness should be in harmony with modern ideas of factory sanitation.

Chromo-lithography.—This growing industry affords employment to a large number of persons, including females and minors. The work is often carried on under unfavorable hygienic conditions as regards light, air space, and ventilation, which is all the more regrettable as some of the processes involve the employment of injurious agents, such as arsenic pigments, chromium, lead and bronze powder. In the room where the prints are made the air is usually charged with vapors of turpentine and benzine.

The lithographic process varies in different establishments; the design or picture may be engraved by means of a diamond or steel needle upon a fine grained sandstone, generally imported from Bavaria, or the drawing may be made upon the stone with a greasy composition. In photo-lithography the printing surface is largely prepared by a photographic process. In all of the processes of chromo-lithography the stone is dampened on those portions of the design which are not to appear in the first printing; this prevents taking the ink or colors. The actual application of colors, and press work, is performed by another set of operatives. According to Leiser[15] in a total membership of 11,807 employees in this industry, in Berlin during 1904, 2002 males and 1840 females, or 32.6 per cent., were reported sick, with 90 deaths; of the sick about 12 per cent. suffered from tuberculosis; 11 per cent. had skin affections, especially obstinate forms of eczema caused by contact with acid dips, bronzing powder, toxic color pigments, impure turpentine, etc.; 10.5 per cent. of the sick suffered from disorders of the digestive system and 19 per cent. of the females were anæmic. There were 356 accidents, mostly contusions of the fingers contracted in press work. Cohen estimates that about 45 per cent. of the lithographers suffered from near sight or other visual defects.

The operation of bronzing may be done by the dry or wet method by hand or machinery. The wet process by inks has not proved satisfactory from the artistic standpoint and machines are rapidly displacing the dry bronzing by hand. The machines print the designs in sizing and the sheets are fed into the bronzing machines from which, however, according to the Massachusetts Report of the State Board of Health,[16] "in spite of their metallic coverings and exhaust ventilation the bronze powder escapes freely into the air. The boys who run the five machines wear handkerchiefs over the nose and mouth. They look pale and unhealthy and all show the characteristic green perspiration due to contact with bronze. The great majority of the employees appear to be healthy." (See also bronzing powder, page 497.)

The danger from lead poisoning is quite pronounced in the manufacture of so-called lithographic transfers intended for decorative purposes on porcelain ware, ornamental glass for windows, art and enameled goods, etc. In this process metallochrome powder, often containing 50 to 60 per cent. of

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white lead, is used. The work of rubbing the powder over the sheets of paper is usually done by females, many of whom show evidence of chronic lead poisoning; indeed it was not until several deaths from acute lead poisoning were reported in this industry that the real cause was discovered. Between 1895 and 1905, 46 cases were reported in Great Britain, and in the latter year 21 cases with 355 days of sickness occurred in one of the German establishments. Several cases were reported by the Illinois Occupational Diseases Commission in 1910. Since that time the dangers have been materially diminished as the "laying on" of the powder is now done on tables covered with a glass case, the hands protected with rubber gloves perform the work through suitable openings, and the dust evolved in the glass case is rapidly removed by exhaust ventilation.[17]

Neisser[18] points out another source of danger in this industry by reporting a case of double cataract in a man who melted over an open flame a color mixture composed of wax, stearin, Venetian soap, asphalt, pitch, turpentine and oil of lavender, mixed in the order named. The affection was attributed to the inflammable character of the vapors evolved during the melting process and the manufacturer has guarded against a recurrence of the evil by providing suitable exhaust ventilation and eye protectors.

Engraving.—The work of engraving upon steel, copper or other material involves not only considerable eye strain, but also, in the absence of suitable work benches, a faulty position which interferes with the respiratory movements. Steel and copper plate engravers are also exposed to mercury, and makers of stamping devices and seals to lead and the fumes of nitric acid. The latter agent is likewise employed in mints in connection with the coinage of money. According to Sommerfeld, cited by Zadek,[19] 73.6 per cent. of all the deaths among this class of workers in Berlin were caused by diseases of the respiratory system, inclusive of 62.1 per cent. from tuberculosis; this is in part accounted for by the fact that so many weaklings engage in this pursuit. Visual defects, neurasthenia, diseases of the digestive system, are also quite common. *Plate printers* who have to look constantly at bright plates, suffer not only from eye strain but occasionally also from conjunctivitis and even retinitis.

Photo Engravers.—Photo-engravers handle benzol for dissolving rubber films, alcohol and ether, in collodium films, strong acetic acid for the removal of films; they are also exposed to gas fumes and ammonium dichromate in sensitizing copper plates, and to ferric chloride and nitric acid fumes in etching. The Report of the Photo-engraver's Union for 1914, cited by Hayhurst,[20] discloses the fact that out of 217 deaths since 1903, 88, or nearly 41 per cent., were due to tuberculosis. During the year 1914, five cases of bichromate and one case of cyanide poisoning were reported.

Metal Etching.—In the etching process of zinc or copper plates nitric acid in the proportion of one part to four of water is used, evolving large volumes of nitrous fumes, which are distinctly injurious, unless the process is

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carried on under hoods equipped with efficient exhaust ventilation. There is also exposure to mercury in some of the processes, and etching on brass involves the use of chloride of mercury.

Neisser[21] reports that in one of the establishments at Nancy chromic and phosphoric acid was employed as a secret process in the etching of zinc plates, causing characteristic chrome ulcers on the hands, arms, neck and face in seven of the 45 persons engaged in the work.

Photography.—Workers in photographic establishments, especially those engaged in the developing process are exposed to bad air and light and also to a number of industrial poisons, such as the bromine, chromium, metol, cyanogen, platinum, vanadium, aniline, and mercurial compounds. They also handle more or less iodine, nitrate of silver and copper sulphate.

In an examination of 40 studios in Chicago by Dr. Karasek[22] platinum paper was found to be the cause of eight cases of poisoning, characterized by "pronounced irritation of the throat and nasal passages, causing violent sneezing and coughing; bronchial irritation, causing such respiratory difficulties as to preclude the use of the paper entirely for some individuals, and irritation upon contact with the skin, causing cracking, bleeding and pain."

Metol poisoning characterized by an erythematous rash of the hands and arms, occasionally involving other parts of the body and giving rise to ulcers, were found in 31 cases in this same study.

The developing process very frequently gives rise to inflammatory conditions of the hands (eczema) ulcers and pigmentation unless protected by rubber gloves.

Chrome compounds and aniline colors are also used in the composition of hectograph inks, and hence are a possible source of danger to the makers and users of the ribbons.

Robinson and Wilson[23] report that in an examination of 1376 employees inclusive of 449 females in the *Printing* and *Publishing Industry* in Cincinnati, only five were found with evidences of tuberculosis, a percentage of 0.36; 17 others were noted as below the normal standard of health. This remarkable low rate of incidence is attributed to the fact that the two establishments where the greater number of examinations were made, were excellent examples of how printing, publishing, and binding industries should be conducted. None of the five cases could be charged to the occupation or bad working conditions. One of the establishments was located in the outskirts of the city with large grounds and excellent sanitary and working conditions. The other large plant, manufactured *Christmas cards*, *calendars*, cards, etc. and employed chiefly girls. The work was clean and apparently without hazard. It is to be hoped that these examples of sanitation in hazardous occupations will be emulated.

One case of tuberculosis was found in an examination of 118 *electrotypers*. The chief hazard reported was the excessive heat and dryness in the plating

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room, the temperature in summer in spite of hoods and exhaust fans was 100 degrees and the relative humidity 35. In winter the temperature was 76 degrees with a relative humidity of 13.

Among 366 workers inclusive of 99 females engaged in *lithographing* not a single case of tuberculosis was found, and only eight could be classified as below the normal standard of health. This favorable showing is attributed by the investigators to the absence of special hazards, commendable sanitary and hygienic conditions, good wages, a full hour for lunch, absence of promiscuous spitting, and absence of noise and the high nervous strain observed in many other industries.

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CHAPTER VI

MINING, QUARRYING AND ALLIED INDUSTRIES

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Air of Mines. Carbon Monoxide. Sulphureted Hydrogen. Marsh Gas. Fire-damp. After-damp. Explosive Conditions in Mines. Diseases of Miners, Tunnel Workers and Quarrymen. Preventive Measures. Stonecutters anad Marble Workers. The Clay Products Industry. The Pottery, Terra-cotta and Fire Clay Product Industry. Sagger Making. Bisque Making. Glaze Mixing. Glaze Dipping. Decorating Department. Lime Burning. Meerschaum. Mica. Cement Workers. Asphalt Workers. Brick and Stone Masons. Plasterers. Paperhangers. Emery and Corundum. Emery and Sandpaper Making. Diamond Cutters and Polishers of Precious Stones. The Glass Industry. Glass Blowing and Polishing. Mineral Wool. Workers in Röntgen Tubes and Röntographers. Radium. Preventive Measures.

Miners and Quarrymen.—Mining, even under the best conditions, is one of the most laborious and dangerous occupations, and according to the German statistics cited by Lindemann[1] it occupies the seventh place in reference to accident occurrence. The liability to accidents is very much greater than in other occupations, but has been reduced by efficient safeguards. The accident fatality rate among German miners has gradually decreased from 3.18 per 1000 in 1879 to 2.25 per 1000 in 1910, while the rate among American coal miners has unfortunately increased from 2.32 per 1000 in 1886 to 3.30 in 1914, as compared with 0.96 for India, 1.02 for Belgium, 1.04 for Austria, 1.69 for France, 1.36 for Great Britain, 2.11 for Germany, and 2.92 for Japan. Professor Holmes ascribes the greater frequency of accidents in the mines of this country to several factors, such as the rapid development and still unorganized condition of the industry, to unskilled foreign labor of a migratory disposition unable to understand the language, to difference in details in the rules and regulations adopted by different mining companies, and last but not least to the sharp competition, especially in coal mining, as a result of which the average American coal miner produces three times as much coal each working day as is produced daily by each miner in Great Britain. On the basis of the number of men killed for each million of tons mined, the death rate for the period of 1901–1910, as given by Holmes, was 5.83 for the United States, 4.40 for Great Britain, 5.05 for Austria, 5.56 for Belgium, 7.55 for Germany, 7.19 for France, 9 for India, and 22.71 for Japan. During 1913 among 747,644 wage earners 2785 men were killed in the coal mines. In the same year among 193,088 workers in the metal mines 683 were killed, or a fatality rate of 3.54 per 1000 as compared with 4.19 in 1911. In addition to the fatal

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accidents in metal mining there were 5890 serious injuries, such as fractures of arms, legs or ribs, and other injuries, causing the loss of more than 20 days' work, and 27,081 slight injuries involving disability from 1 to 20 days. The economic loss from all three classes of injuries due to accidents in coal mines, according to the U. S. Bureau of Mines,[1] for 1911 would total $14,142,000; and the loss of 38,853 lives in the coal mines of the U. S. in the 19 years from 1896 to 1914 would amount to over $194,000,000.

The causes of fatal coal mine accidents in the U. S. during 1912 are given by F. W. Horton[2] as follows: Falls of roof (coal, rock, etc.), 41.19 per cent.; falls of coal other than roof coal, 7.58 per cent.; mine cars and locomotives, 15.34 per cent.; gas explosions and burning gas, 6.95 per cent.; coal dust explosions, 1.27 per cent.; explosions of coal dust and gas, 4.53 per cent.; explosives, inclusive of suffocation by gases from explosives, 5.64 per cent.; suffocation from mine gases, 0.42 per cent.; electricity (shock or burns), 3.22 per cent.; animals, 0.30 per cent.; mining machines, 0.59 per cent.; mine fires, burned, suffocated, 0.47 per cent.; other causes, 2.20 per cent., making a total of 89.79 per cent. killed underground. 2.29 per cent. of the total fatal accidents occurred in shafts; about one-half were caused by falling down shafts or slopes and from objects falling down shafts or slopes, breaking of cables and chains, overwinding and other causes. 7.92 per cent. of the fatal accidents occurred on the surface and were caused by mine cars and locomotives, electricity, machinery, boiler explosions, railway cars and locomotives and other causes.

In 275 coal mine accidents in the U. S. up to and including 1912 in which 5 or more men were killed, involving a total loss of 6777 men, 5111 or about 75 per cent. were killed by gas and coal dust explosions, 1082 or over 15 per cent. lost their lives by mine fires, 159 were killed by explosives, 123 by inrush of water, 105 by falls of roof and coal, 26 by mine cars and locomotives, 89 by shaft accidents and 82 by other causes.

According to Fay,[3] about two-thirds of the total deaths and injuries that occurred in or about metal mines in 1913 were caused as follows: Of the fatal aciddents 36.16 per cent. were due to falls of ore or rocks from roof, wall or bank; 13.04 per cent. to explosives; 12.31 per cent. to falling down slope, shaft or winze, and 11.42 per cent. to haulage systems.

Of the serious accidents 24.12 per cent. are due by falls of roof, wall or bank; 17.22 per cent. to car and haulage systems; 9.43 per cent. to machinery, and 12.69 per cent. to timber and hand tools. Of the slight accidents, 22.65 per cent. are due to falls of roof, wall or bank; 13.27 per cent. to car and haulage systems; 8.83 per cent. to machinery, and 15.78 per cent. to timber and hand tools.

Air of Mines.—The air of mines differs in composition from the atmospheres in this, that there is always an excess of carbon dioxide, also known by miners as black-damp or choke-damp.

It is a product of decomposition and combustion and of respiration of

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men and animals, and is naturally increased by the process of breathing, by the burning of miners' lamps and by blasting operations, and its presence in dangerous volumes extinguishes a flame. Carbon dioxide is not itself a toxic gas, but will cause asphyxia when present in sufficient volume to interfere with the atmospheric oxygen in the performance of its function. Most authorities agree that an atmosphere containing 4 per cent. of CO_2 and about 16 per cent. of oxygen cannot support life for any length of time, since the blood cannot get enough oxygen for the needs of the cells, nor can the blood rid itself of its CO_2, because the tension of CO_2 in the atmosphere exceeds that of the CO_2 in the blood. Under such conditions the blood corpuscles cannot excrete it and asphyxia results. In addition to the gases normally present in the atmosphere the following should be mentioned as sources of danger in mines.

Carbon monoxide, CO, also known as "white-damp," when present in the air of mines is the product of slow or incomplete combustion of carbon, and is chiefly produced during blasting operations and by "gob fires" where the air is limited. For symptoms of poisoning see pages 59 and 726.

Apart from being a profound blood poison, its presence in fire-damp mixtures tends to widen the explosive effects of fire-damp.

The danger of carbon monoxide even in small volume is so great that Haldane recommends the use of birds and mice for the purpose of detecting harmful quantities of this gas, as a protection to exploring and rescue parties in mines. The U. S. Bureau of Mines[4] found that a canary bird collapsed in the presence of between 0.20 and 0.30 per cent. of carbon monoxide, while the members of the exploring party experienced no symptoms of distress. The bird quickly revived when placed in better air. The Bureau of Mines strongly urges general adoption of the bird test in this country.

Sulphureted hydrogen, H_2S, may be found occasionally as an occluded gas in coal seams, and in mines is usually the product of decomposition of pyrites in the presence of moisture. It is a blood poison, as it takes away the oxyhemoglobin, and is generally recognized by miners as "stink-damp" on account of the odor resembling that of rotten eggs. When mixed with seven times its volume of air it is violently explosive.

Marsh gas, CH_4, also known as light carbureted hydrogen and methane, is the product of decomposition of carbonaceous matter, which has taken place with the exclusion of air and in the presence of water. It is one of the characteristic occluded gases of coal mines, and is not observed in metalliferous mines. Olefiant gas, C_2H_4, and ethane, C_2H_6, are known as heavy hydrocarbon gases and are likewise the product of the decomposition of carbonaceous matter but in the absence of water, hence they contain a higher percentage of carbon than marsh gas. Olefiant gas and ethane are rarely found under normal conditions in coal mines, and if present they are the result of a mine fire or the intrusion of gas from a natural gas well.

Fire-damp.—The constituents of fire-damp are methane and air. It is of special importance as marsh gas, in the proportion of 1 to 13 of air, forms

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an explosive mixture, which reaches its maximum violence when the proportion of carbureted hydrogen is 1 to 9½.

After-damp.—This term refers to the gaseous mixture in mines after an explosion of gas. The chief products of a fire-damp explosion are carbon dioxide, watery vapor, nitrogen and CO. When a large body of gas has been exploded and the air of the mine does not furnish sufficient oxygen for the complete combustion of fire-damp, a large amount of carbon monoxide may be present in the after-damp. For like reasons we may expect an abnormal amount of carbon monoxide in the air of mines after a coal dust explosion or during blasting operations.[5]

In addition to carbon monoxide, the nitrous fumes given off during the explosion of dynamite, gelignite, roburite, blasting gelatine, etc., doubtless play an important rôle in the development of acute respiratory diseases and other affections. The undue prevalence of pneumonia among the workers in some of the mines, both here and in South Africa, may in part be due to the fact that exposure to nitrous gases, apart from engendering pulmonary congestion, also produces methemoglobin. It has been noted by Dr. Huntington, cited by Dr. Ebright,[6] that men engaged in blasting operations are inclined to be irritable and pugnacious and that it is unsafe to pick a quarrel with them. Dr. Ebright attributes this to the fact that alcohol enhances the toxic effects of nitroglycerine, and in support cites a case in which acute mania with homicidal impulses developed.

Explosive Conditions in Mines.—Among the causes which render explosive conditions in mines possible are the following: 1. Defective ventilation; (2) sudden increase of explosive gases from falls of roof; (3) dry coal dust suspended in the air; (4) pressure due to a heavy blast or concussion of the air; (5) rapid succession of shots in close workings; (6) accidental discharge of an explosive in a dusty atmosphere. The exciting cause may be ignition of fire-damp, but much more frequently it is the ignition of fine coal dust, caused by a blown-out shot.

Preventive Measures.—For the prevention of fire-damp explosions, safety lamps, electric lights, copious ventilation and safety rules have been found most efficient. Copious ventilation, on the other hand, has proved a source of danger by stirring up fine coal dust and carrying it to the roofs, sides, bars and props of the mines, and thus greatly increasing the dangers from coal dust explosions.

This danger is generally recognized and recommendations for the prevention of such catastrophes have been made. Garforth, cited by Oliver,[7] suggests fixing the dust (1) by water spraying under high pressure; (2) by keeping the roadways free from dust for certain lengths, thus creating dustless zones; (3) covering or diluting the coal dust with stone dust. Ascher and others recommend spraying with calcium and magnesium chlorides mixed with silicate of soda, glycerine, water, etc. Thornton advocates spraying with soapy water; others advocate the removal of coal dust by

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vacuum cleaners. So far the creation of dustless zones and the use of stone dust, urged by Garforth, appear to give promise of the most satisfactory results. Water spraying was followed by an increase in the number of cases of hook-worm infection among the German miners in the Valley of the Ruhr. Prof. Holmes, Director of the Bureau of Mines,[8] finds that there is little or no danger from coal dust explosion in the spring, summer and early autumn months, as the warm air admitted from without, upon being cooled by contact with the cool walls within, deposits its moisture and keeps up a continuous sweating. During the cold winter months it has been found that the introduction of exhaust steam from the mine power plant in sufficient quantities to raise the temperature of the entering air, and at the same time to supply it with moisture, offers a simple safeguard. Prof. Holmes fully realizes that this method, as well as spraying, is likely to favor the development and spread of the hook-worm disease, and also to create less favorable working conditions as regards humidity and temperature. But happily neither of these evils has appeared after an experience of nearly 5 years.

Diseases of Miners, Tunnel Workers and Quarrymen.—Lindemann's[9] German statistics for 1911, based upon a membership of 357,321, show that diseases of the digestive organs lead the list in the order of frequency with 39,725 cases or 11.1 per cent.; (2) diseases of the respiratory organs, 28,954 or 81 per cent.; (3) neuralgia, 33,991 or 0.94 per cent.; (4) acute articular rheumatism, 3482 or 0.97 per cent.; (5) muscular rheumatism, 30,008 or 0.83 per cent.; (6) pneumonia, 1774 or 0.49 per cent.; (7) nystagmus, 1356 or 0.37 per cent.; (8) hook-worm disease, 1166 or 0.32 per cent.; (9) emphysema and asthma, 689 or 0.19 per cent.; (10) heart disease, 449 or 0.12 per cent.

Miners and tunnel workers may also be subject to compressed-air disease, and Dr. G. W. Johnson[10] has reported 42 cases of gas poisoning caused by the fumes of a petroleum motor engine, used in the construction of the Montreal tunnel.

Affections known as "beat hand," "beat knee," "beat elbow" are primarily due to friction of tools or contact with hard ground, etc., causing callosities of the skin beneath which infection may occur, giving rise to subcutaneous cellulitis of the hand, over the patella and elbow. Inflammation of the synovial lining of the wrist-joint and tendon sheaths is not uncommon.

The undue prevalence of diseases of the digestive organs has no special connection with the occupation, except in so far as it tends to the consumption of cold victuals and of excessive quantities of cold water when the body is overheated.

Nieszytka[11] mentions mechanical pressure of the tool upon the stomach and liver, the ingestion of dust, the inhalation of sulphureted hydrogen and carbon monoxide, and the abuse of alcohol as possible factors, and that prohibition of the use of alcoholic beverages during working hours, especially in the clay and stone quarries, has had a beneficial effect. The respiratory diseases, however, are greatly influenced by the character and amount of dust,

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injurious gases, heat and humidity, abrupt changes in temperature, drafts, overwork, etc. The gases in the after-damp, especially carbon monoxide and nitrous fumes, doubtless play an important rôle in the causation of acute pulmonary affections. Workers in quarries, on account of the additional exposure to the elements, are much more liable to pneumonia than miners. The majority of chronic respiratory diseases begin as catarrhal affections of the upper air passages—bronchial catarrh—which may terminate in or be complicated sooner or later with pneumoconiosis (see p. 218), emphysema and tuberculosis.

The character of dust doubtless plays a more important rôle than the amount. In no other way can we explain the comparative innocuity of coal dust, the particles of which are quite free from sharp points and corners. Dr. Ogle, in the 45th Annual Report of the Registrar General, has shown that coal miners stand at the head of the list as regards comparative immunity from phthisis and other lung diseases in dust-inhaling occupations. On the other hand, the Cornish tin miners, who are exposed to the inhalation of a sharp angular and most irritant type of dust, furnish the largest number of cases. This difference in the character of the dust is also strikingly illustrated by our American statistics, which show a mortality from tuberculosis and pneumonia of 25.8 per cent. among coal miners, compared with 31.63 per cent. among metal miners and 43.5 per cent. among copper miners. The observations among the gold quartz miners of South Africa has already been referred to. Dr. A. J. Lanza of the U. S. P. H. S. in a recent address before the American Public Health Association directed attention to the unusual amount of pulmonary diseases among the zinc miners of the Joplin district as compared with similar miners in southeastern Missouri, and attributes the difference to the presence of large amounts of flint dust, which has a silica content of over 95 per cent. Nieszytka[12] states that salt and sulphur mining, and all metal mining involving exposure to dust containing lead, predispose to tuberculosis; on the other hand, Caryophilis reports only five cases of tuberculosis among 1932 Greek sulphur miners. Nieszytka reports that 76.5 per cent. of all the deaths among the sandstone workers at Hannover are caused by tuberculosis and that their average age is only 35.1 years. On the other hand, he cites Grab's statistics to show that in limestone workers tuberculosis is the cause of death in only 7.5 per cent. of the total mortality, with a tuberculosis morbidity rate according to Schlokow of only 2.2 per 1000. Koelsch[13] confirms Grab's statistics with reference to the lime and cement industry and cites the statistics of a German Benefit Society to show that among 400 workers, in a German plaster-of-Paris establishment, no cases of tuberculosis occurred during a period of 17 years, and that of 40,824 deaths from tuberculosis analyzed by Fisac in Spain, only 17 or 0.41 per 1000 occurred in lime or gypsum workers. Selkirk,[14] of our own country, was also unable to find a single case of phthisis among lime workers, nor could he learn of any worker in limekilns having died from this disease. He suggests that the

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workingman predisposed to tuberculosis might turn his attention to lime and cement working as an occupation, and even hints at the organization of lime works as a curative for a tuberculosis colony. All such statements deserve painstaking investigations. While it must be conceded that lime and plaster of Paris belong to the less irritant kinds of dust, in our present state of knowledge it cannot be claimed that their inhalation prevents tuberculosis or is in any way a curative agent.

The tuberculosis death rate among the workers in slate quarries in Thuringia amounts to 43.1 per cent. and even to 64.3 per cent. among the slate-pencil makers, and this in spite of the fact that dust production is not specially excessive. It is also high among the slate workers of Wales. Oliver suggests that the bad housing and living conditions are important factors.

The quarrymen and stonebreakers of the Leipsic Sick Benefit Society have the third highest morbidity rate, viz., 1496 days per 100 workers, and the mortality hazard is 0.88 per cent.

The undue prevalence of rheumatic and neuralgic affections is doubtless influenced by unfavorable working conditions, such as heat, humidity, sudden changes, stooped positions, contact with wet ground, scant clothing, etc. Hard work, articular rheumatism and alterations in the lung tissue, such as pneumoconiosis, emphysema, etc., naturally favor the development of heart lesions.

The cause of nystagmus is still somewhat obscure. Butler[15] believes that chronic carbon monoxide poisoning is one of the chief factors. Lindemann's experience leads him to conclude that the muscular strain incident to an abnormal position of the eyeball, and the glare of the miner's lamp, are more important factors than defective light, carbon monoxide gas, and other suggested causes. He believes that chronic inflammatory conditions of the eyes, opacity of the cornea, visual defects, anæmia, general weakness and alcoholism also favor the development of nystagmus, which, while affecting between 3 and 4 per 1000 miners, is rarely of sufficient gravity to prevent recognition of a 2 to 3 per cent. fire-damp in the flame of a benzine safety lamp.

Hook-worm disease deserves special attention, as the conditions in mines, with special reference to absence of privies, temperature, humidity, exposed portions of the skin, and the eating of food with unclean hands favor the development and spread of the disease. In 1904 hook-worm infection was noted in 13,861 Westphalian miners; in 1911 there were still 1166 cases or 3.26 per 1000. In the meantime an energetic campaign had been waged against the spread of the infection by the erection of transportable privies for the prevention of soil pollution, disinfection of stools by means of milk of lime, and by the administration of thymol for the destruction of the parasite in infected individuals.

The carriers of infection are not permitted to resume work in the mines

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until found free from worms and eggs for a period of 2 weeks after cessation of medication; a reëxamination is made at the expiration of 6 weeks.

Dr. Lanza, in the address on the health hazards of the metal mining industry already referred to, states that hook-worm disease has been found among miners in southern California; other mining sections appear to be as yet free from the infection. Amebic dysentery has been observed in some of the Arizona mining camps, following the importation of Mexican labor, and he fears that hook-worm may also gain a foothold there.

Anæmia has always been quite prevalent among miners and at one time the belief that chronic carbon monoxide poisoning and absence of sunlight played an important rôle in the causation of anæmia montana appeared justified. It is highly probable, however, that hook-worm infection and chronic pulmonary diseases are the most important factors. For caisson disease in tunnel workers see page 188.

Preventive Measures.—Commendable progress is being made in the substitution of machine labor for hand labor. This, together with reasonable hours of work, diminution in dust production and its removal by exhaust ventilation, will materially aid not only in the prevention of diseases of the respiratory and circulatory organs, but also in the prevention of accidents from explosions of gases and coal dust. The general use of permissible explosives should be insisted upon. The question of adequate ventilation is of special importance. According to Dr. Lanza, in France the law limits the regular working temperature to 96° dry and 86° wet bulb. In Victoria and New Zealand 80° wet bulb is the limit. In Germany when the wet bulb is above 80° the shift is reduced from 8 to 6 hours. The undue prevalence of rheumatic and neuralgic affections can be materially checked by suitable work suits and avoidance of abrupt changes in temperature, and by shower baths and change of clothing upon cessation of work. Men engaged in quarries should be provided with suitable shelter during inclement weather; the employment of respirators and eye protectors is clearly indicated. No effort should be spared to improve the housing and living conditions of miners and quarrymen, whose natural powers of physical endurance are severely taxed by so many injurious factors connected with their work.

Stonecutters and Marble Workers.—These occupations have from time immemorial been regarded as inimical to health and even Ramazzini, in the first book on Occupational Diseases, calls attention to the fact that the inhalation of the dust incident to hewing, cutting and polishing of marble or of stone produces a troublesome cough, and that a goodly number of the operatives become asthmatic and consumptive. We know now that the inhalation of mineral dust develops sooner or later pneumoconiosis which may eventuate in pulmonary tuberculosis. It is generally held that the liability to diseases of the respiratory passages is less in the case of paving stonecutters and slate splitters, and in the sawing, grinding, polishing and lathe work which can be conducted by the wet process, than in the case of

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monument or custom work, and particularly in the surfacing, carving and cutting with pneumatic tools. The greatest amount of dust is evolved by the surfacing machines which are operated with compressed air. Of the various tools employed, the bushing hammer creates the finest dust. Unfortunately, work with pneumatic tools cannot be done by the wet process, as the pasty material created by a mixture of water and dust clogs up the tools. This work is usually done in large open sheds or in the yards, but even under such conditions the men are exposed to clouds of dust. The sawing of granite and marble into slabs, turning in lathes, and the final polishing can be conducted by the wet process; soapstone sawing and cutting for joints is frequently done dry and is attended with exposure to considerable dust.

The following is a condensed extract of Mr. W. C. Day's[16] description of the polishing process: The slabs of stone to be polished are placed upon a rubbing bed which consists of a circular cast-iron plate from 8 to 15 ft. in diameter with a circular opening about 18 in. in the center, and mounted upon a running gear so that it may revolve in a horizontal plane. An abrading material such as sand sometimes mixed with "chilled shot" or crushed steel, with a constant supply of water, is fed upon the plate. When the process has gone far enough, the slabs are removed to the emery bed, which is similar to the one described, fine emery being used for abrasion. The slabs are next rubbed down by hand with a fine, evenly grained sandstone, known as "Scotch hone," and smoothed off with pumice stone. The final polish is put on by placing the slabs upon a buffing bed, which is similar to the rubbing bed but covered with a thick specially prepared felt. A small amount of putty powder is fed to give a high polish. The hand process consists in grinding on the rubbing bed as before, and then rubbing down by hand, successively with Nova Scotia "blue stone," "red stone," "Scotch hone" and pumice stone, after which it is glossed with putty powder, or in case of cheaper "onyxes" and common marbles, with a mixture of two parts of oxalic acid and one part of tin oxide.

Of the various kinds of stone dust some are doubtless more injurious than others. Reference has already been made to the excessive death rates from tuberculosis among workers in sandstone and the low rates in limestone workers in Germany. Hoffman[17] cites two observations in this country which apparently corroborate the German statistics in this respect. According to an observer of conditions in the sandstone industry, sandstone cutters seldom live to be 50 years of age, and nearly all of them die of lung disease due to the inhalation of mineral dust. A physician of long residence in the New Bedford Indiana limestone field states that he has not observed very serious consequences to result from employment in that industry, but as justly remarked by Hoffman "all such observations are subject to serious error in the absence of trustworthy statistical data for a period of years."

The Inspectors of the State Board of Health of Massachusetts[18] regard granite dust more injurious than marble and soapstone the least of all; some

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varieties of granite yield a much finer dust than others on account of differences of texture. It should be remembered, however, that cutting of all varieties of stone involves exposure to dust, and that the character of the dust, the environments under which the work is performed, the social conditions, such as housing, etc., doubtless influence to a great extent the liability to pulmonary affections.

A collective investigation cited by Roth[19] shows that of every 100 deaths among stonecutters, polishers, etc., 86 were due to diseases of the lungs, inclusive of 55 deaths from tuberculosis.

Of 2013 stonecutters examined by Sommerfeld[19] 19.7 per cent. were afflicted with tuberculosis, 17.98 per cent. with other diseases of the lungs, and nearly all had a chronic catarrh of the throat or larynx. According to the report of the State Board of Health of Massachusetts[20] of 343 deaths which occurred in the city of Quincy among stonecutters during a period of 16 years 41.4 per cent. were caused by consumption, 12 per cent. were due to other diseases of the lungs; 12.8 per cent. to diseases of the heart; 7 per cent. to violence; and 26.8 per cent. to all other causes.

In analyzing the statistics of the towns in the State of Vermont, where most of the granite and marble industry is carried on, the writer found[21] that Barre, Montpelier, Rutland, Proctor, Dorset, Hardwick, Bethel and Ryegate, with a combined population of 34,889, had a tuberculosis death rate of 2.2 per 1000 of population, against a rate of 1.3 for the entire State.

Hoffman's[22] statistics based upon 534 deaths among stonecutters from all causes, shows that the principal cause of death was tuberculosis, amounting to 47.8 per cent. at the ages between 25 and 44 years, and at ages between 45–62 to 32.3 per cent. of the mortality from all causes. According to the recent Ohio Survey by Hayhurst, of 163 deaths between 1910 and 1912, among the marble and stonecutters, 27.61 per cent. were caused by pulmonary tuberculosis, as compared with 7.13 per cent. in persons engaged in agricultural pursuits. The Leipsic statistics show a sickness rate for marble cutters of 1294 days for 100 members per annum and a mortality hazard of 1.27 per cent.

Millstone cutting has always been regarded as a dangerous occupation, with special reference to pulmonary diseases. This is due to the fact that on account of the very hard character of buhr-stone, which is composed of a variety of quartz, there is an unusual wear and tear of the steel tools with which the stone is dressed, and the men are exposed to a combination of steel and mineral dust. Oliver[23] found very few old men in this industry, and states that buhr-stone workers are frequently intemperate. The same may be said of stone workers in general, who attribute the habit to the dryness of the throat caused by the lodgment of dust. It must be conceded that all dust-producing occupations are predisposing factors to the alcoholic habit, especially when the employer makes no attempt to furnish pure water, and

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when the regulations for the prevention and removal of dust are totally ignored.

Apart from the danger of dust inhalation the liability to accidents is not inconsiderable in all stonecutting operations, injuries to the eyes from flying chips and fragments of steel from implements are especially common. Rheumatic affections and colds, as a result of exposure are also quite common. The work is very laborious and none but able-bodied men should enter the trade.

Preventive Measures.—All the precautions mentioned in the preceding section for the protection of quarrymen, such as wet processes, general cleanliness and copious downward exhaust ventilation, use of goggles and respirators are applicable for stonecutters.

The Clay Products Industry.—The brick and tile industry includes the manufacture of building bricks, fancy and ornamental bricks, vitrified paving brick, drain tile and sewer pipe. During the last U. S. census 76,528 persons, including 81 women and 1270 persons under 16 years of age, were employed in this industry.

The preliminary work, such as grinding and mixing of clay involves exposure to dust, also to the elements and to wet processes, while in the vicinity of the kilns, there is more or less exposure to excessive heat, sudden changes in temperature, drafts and to the inhalation of CO, CO_2, and of sulphurous acid.

Instances of carbon monoxide poisoning have occurred in men entering the kilns for the purpose of filling or emptying, also during the burning process, by the escape of gas from defective kilns and flues. Rambousek cites the case of a worker who was about to fill a kiln, and upon opening the furnace door was overcome by large volumes of CO, which proved fatal, in spite of efforts at resuscitation.

The German statistics, cited by Gottschalk[24] show that nearly 21 per cent. of all the sickness is caused by muscular rheumatism, mostly lumbago which is frequently accompanied by sciatica and other neuralgia. Pneumonia and pleurisy contributed 5.4 per cent., and colds and catarrhal affections of the respiratory organs 18.1 per cent. of the general morbidity. The catarrhal affections are to a considerable extent induced by inhalation of dust, and German authors have found a sufficient number of "clay lungs" to justify the term aluminosis. During the winter months influenza contributes an additional 10 per cent. to the morbidity rate. It is evident that the undue prevalence of diseases of the respiratory organs paves the way for tuberculosis. 12.3 per cent. of the morbidity was caused by diseases of the digestive system, probably due to sudden changes in temperature, improper food, and the pernicious habit of cold drinks while the body is overheated.

On account of soil pollution caused by primitive habits at the clay banks there is also a certain amount of danger from infection with the hook-worm, the small intestinal round worm and typhoid organisms. Varicose veins and ulcers are not uncommon.

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Callosities of the hands, as a result of hard work, and eczema of the hands and wrists from exposure to dust, dirt and moisture are frequently observed. Most of the glazing is done by the addition of salt. The danger from lead poisoning in the manufacture of fancy ornamental and enameled bricks, due to the employment of glazes containing lead, will be referred to in connection with the pottery industry.

Preventive Measures.—The hygiene of this industry has been very much neglected and this is especially true with reference to clean, decent quarters and sanitary conveniences for unmarried employees, and pure drinking water. Persons engaged in wet work should wear rubber boots and aprons. Dust production should be reduced to a minimum. The men employed about the brick kilns should wear suitable clothing and protect their eyes from exposure to intense heat and light, and should receive instructions how to avoid injurious gases. Washing facilities before each meal, and facilities for a full bath and change of clothing upon cessation of work, should be provided.

The Pottery, Terra Cotta and Fire Clay Product Industry.—The chief products of this industry are stoneware, earthenware, porcelain ware, firebrick and terra-cotta products.

During the last U. S. census there were 61,922 persons engaged in this industry of whom 56,168 were wage earners, inclusive of 7000 females and 816 children under 16 years of age.

The ordinary stoneware and earthenware is usually made of common clay. For the better grades of goods, china clay or calcined bone are added; and for the still higher grades of porcelain a mixture composed of very fine clay, calcined bone and feldspar is used. The most expensive ware like Limoge porcelain is made from very fine white clay peculiar to that section. The removal of the different clays from stock bins, the crushing, grinding, mixing and sifting processes, and especially the process known as "flint dust making" are attended with the evolution of large amounts of dust.

There is also more or less exposure to plaster-of-Paris dust in the manufacture of moulds for forms for china, art ware, etc.

The chief factors inimical to health in the so-called "slip houses" are dust, heat, fatigue and dampness from faulty floor drainage, leaky vats and slip-containing machines and the presence of wet clay under foot.

Sagger Making.—This process which consists in shaping, repairing, baking and finishing of clay containers in which the pottery ware is subsequently baked, and involves similar hazards, and also the risk of lead poisoning, from the red lead solution with which the inside of the saggers are painted. The Ohio Survey disclosed four cases of lead poisoning among 20 workers in this branch.

Bisque Making.—In this department the clays previously prepared in the "slip" house are shaped on plaster-of-Paris moulds and potter's wheels. In tile and procelain factories this work is done by machines in so-called

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press rooms. After the ware is shaped, it is carried, usually by boys, into the drying kilns, located in the rear of the workers. This process involves exposure to dust, heat and fumes from the baking ovens, unless efficient exhaust ventilation is provided.

Glaze Mixing.—In order to render clay products impervious, and to secure a polished surface, they are dipped into a liquid glaze containing finely ground clay and flint and more or less white carbonate or red oxide of lead. Some of the glazes do not contain lead, but most of them do, and when used there is naturally considerable exposure to lead dust. Of 865 cases of lead poisoning which occurred between 1901 and 1909, in the potteries of Great Britain, 788 were contracted in glaze processes, 51 in decorative processes, and 26 in unclassified processes.[25]

Glaze Dipping.—After drying or bisqueting, and sometimes even in the green state, the ware is dipped into a glaze solution, or the solution is applied by means of a brush or sponge. The glazes used for porcelain, stoneware, and some tiles contain usually no lead. The colored glazes are rich in lead and the majority of glazes contain lead in an unfritted form, which is deplorable as "fritted" lead, which is a fixed compound with borax and silica, is less soluble and hence less dangerous. The dipping is generally done by hand, although recently dipping machines specially adapted for tiles and other regular pieces are employed and have reduced the handling of glazes very greatly, and practically obviated the necessity of "fettling" and cleaning of the glaze from the edge of the tile. The process of "fettling" consists in the removal of superfluous glaze from the ware prior to placing it in the glost kiln, but refers more especially to the removal of little projections by means of a small steel knife, and other finishing processes, such as rubbing, sanding and dressing after the ware leaves the kiln.

After glazing, the ware is placed in saggers and fired. Ordinary terra-cotta or stoneware is dipped and fired but once, but most of the other ware is fired twice. In some branches the glaze is applied to already partly glazed ware in the form of lead enamel dust, by means of a cotton pad; this method, known as "ground laying," is dangerous and has been to a great extent replaced by the aerographing process. For roof tiles and bricks the glaze, if used at all, is usually poured on, or applied by means of a brush. The last two methods are sloppy and dangerous because the liquid is liable to splash over the hands, clothing, floor, etc. Kaup[26] reports that in some roof-tiling establishments nearly all of the workers suffer from lead poisoning.

The work in or about the kilns involves exposure to excessive heat, coal gas, dust, abrupt changes in temperature, and lead poisoning. The Ohio Survey discloses about 35 cases of lead poisoning among the 1220 workers around the bisque and glost kilns. The chief risk came from handling the ware upon which the glaze had just been dried and from which it could be wiped off like flour; in some instances, especially in art and colored ware, the dust contained 50 per cent. of lead.

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Decorating Department.—The decorative processes may be carried on before or after the glaze is applied and in some instances the glaze itself is colored. So for example, Rockingham ware is glazed with a dip containing manganese, which results in a brownish or plum colored finish, and jet ware is dipped in a glaze containing cobalt which imparts the jet black finish. Since cobalt is obtained from arsenical ores there is some danger from arsenical poisoning. Most of the decorating processes are carried on by females and weaklings and consist in the laying on of lithographic transfers by means of sizing with turpentine, stamping on impressions, "lining" with gold stripes and gilding, color spraying, hand painting, etc. The employment of lead containing pigments, such as chrome yellow, nitrate of lead or red lead, whether applied by brush, the aerograph or decalcomania, naturally involves exposure to lead poisoning. The Ohio Survey disclosed three cases of lead poisoning, one suspected case of arsenic poisoning and one case from benzine varnish poisoning.

While cases of plumbism in decorative work are now less frequent, the sedentary habits and unfavorable working conditions predispose to neurasthenia, diseases of the respiratory and digestive organs, ocular defects aad inflammatory conditions of the eyes from exposure to turpentine. The danger from dust inhalation in the ceramic industry is plainly revealed by the undue prevalence of respiratory diseases, which according to Holitscher's[27] German statistics were the cause of death in 72.8 per cent. of the potters, and in 62.4 per cent. of the decorators. Hoffman's American Industrial statistics show that 47.9 per cent. of pottery workers perish from respiratory diseases, inclusive of 33.1 per cent. from tuberculosis. Rheumatic and neuralgic affections and obstinate forms of eczema are not uncommon. Cases of lead poisoning are still unduly prevalent, especially in this country, and in the smaller establishments and home industries of Europe. Chyzer,[28] cited by Rambousek, refers to the conditions in Hungary where members of the family contract the disease from the dust in the living rooms, which was found to contain from 0.5 to 8.7 per cent. of lead. The degree of danger depends upon the amount of lead used, which varies from 10 to 24 per cent. in the glaze fluxes, and from 60 to 80 per cent. in the enamel colors for decorative purposes.

Oliver[29] and Thorpe, who investigated the subject of lead poisoning in the potteries of Great Britain, came to the conclusion, as early as 1899, that by far the greater amount of earthenware can be glazed without lead, and that in certain branches of the industry, in which lead is indispensable, it should be used in the form of a fritted double silicate. It has been found that the danger can be materially reduced by using only 8 per cent. of carbonate of lead in the form of a "double fritted silicate" instead of the older method, in which from 13–24 per cent. of lead carbonate was employed. The English authorities determined that no glaze should be used which upon shaking for an hour with 1000 times its weight of a 0.25 per cent. watery solution of hydrochloric acid revealed more than 5 per cent. of its dry weight of lead monoxide. The

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fruits of this splendid work are shown by the fact that in 1898 Great Britain had still 457 cases of lead poisoning among pottery workers, and in 1908 only 58 cases. Unfortunately, we have not profited by this lesson to any considerable extent, for Dr. Alice Hamilton's[30] investigations show that while Great Britain in 1910 had only 77 cases in a force of 6865 workers, in 1911 we had 144 cases in a force of 1500. Nothing should be permitted to stop the campaign for leadless glazes, and since it has been shown, as pointed out by Oliver,[31] that the enameled bricks used in Babylon 522 B. C. and the decorative tiles in Egypt, did not contain lead, it is hoped that chemistry and technical skill will sooner or later solve the problem.

Preventive Measures.—While much of the potter's work is with moist materials, it is evident that the handling of all dry and fired products, and the grinding and mixing of lead compounds of glazes, involves exposure to dust, which should be reduced to a minimum by moist methods. Exhaust ventilation is important in all dusty processes such as fettling and pressing of tiles, bedding and flinting, brushing and scouring of biscuit ware. Exhaust drafts are especially needed in the dusty lead processes, such as mixing and grinding of lead compounds, aerographing, color dusting, and in ware cleaning. Absolute cleanliness of the workshops, cement or impervious floors, frequently washed, proper air-space, good general ventilation, and special lunch rooms are important. Suitable work suits and caps, ablutions before meals, baths and change of clothing upon cessation of work, and monthly medical inspection of workers exposed to lead processes should be insisted upon.

Lime Burning.—The risks incident to the quarrying of limestone have been referred to and also some interesting statistical data in reference to the comparative danger of lime, plaster of Paris and other mineral dusts have been presented. Rössle's[32] recent investigations tend to show that workers in the lime, cement, procelain and glass industries have relatively favorable mortality rates from tuberculosis. The kilns are generally loaded from a car, operated by cable from the top of the kiln platform, and the dumping involves exposure to dust and more or less CO_2 from the open kilns. During the roasting process there is exposure to excessive heat and escaping gases, while the unloading from the false bottom of the kiln and the subsequent grinding and packing of the lime, unless a good suction system is in operation, are naturally very dusty operations. As a result, inflammatory affections of the skin, eyes and of the upper air passages are not infrequent.

Meerschaum, also known as sepiolite, is a soft white hydrous magnesium clay, containing silica, and when dry it will float on water. It is chiefly used for carving tobacco pipes and cigar holders and the dust, like that of talc and soapstone, is a frequent cause of chronic diseases of the respiratory passages.

Mica.—This is a mineral of widely varying chemical composition, but is essentially composed of silicates of aluminum and an alkali, such as potassium, sodium or lithium. The mineral splits easily into thin flexible colorless trans-

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parent plates or scales, known as isinglass, and is used for a number of purposes where glass could not resist the effects of heat. In the powdered form it is employed in the manufacture of a giant powder, and on account of the glistering character of the dust it is largely used for decorative purposes, chiefly in the manufacture of wall paper, illuminated designs, postal cards, etc., and is doubtless a frequent cause of inflammatory conditions of the eyes and air passages.

Cement Workers.—The manufacture of Portland cement varies in different countries. The raw materials are clay with flint and lime. In England where chalk is plentiful this is used in connection with some material containing silica and alumina, such as selected clay or river mud. The raw materials are mixed in certain proportions and ground together to the consistency of liquid mud. The excess of moisture is driven off by heat, and the residue is dried in ovens at a high temperature. The preliminary process, while sloppy, is not dusty work, but the subsequent grinding between rollers and the sacking and packing of the product is an extremely dusty process. According to Koelsch[33] and other German authors catarrhal affections and diseases of the respiratory organs constitute from 30 to 40 per cent. of all the sickness. Diseases of the eyes, and ears, impacted ear wax, ulceration of the nose with perforation of the nasal septum, and eczema, or cement itch, are also quite common. Hoffman's Industrial Insurance statistics show that 47.7 per cent. of the cement workers in this country perish from diseases of the air passages, inclusive of 19.3 per cent. from tuberculosis. The Leipsic Sick Benefit Society records indicate that cement mixers and hod carriers have a very high morbidity rate, viz., 1358 days of sickness per annum per 100 members.

Preventive Measures.—Wittgen[34] has made a gratifying report upon the results attained in German cement works by removal of dust. He points out that respiratory diseases have been reduced fully one-third within a period of 5 years after the installation of exhaust ventilation, and the number of days lost by sickness has been reduced from 2742 to 812 days per annum, which means a decided improvement in the general health of the operatives.

Asphalt Workers.—*Asphaltum*, also known as mineral pitch or hard bitumen, is an amorphous brownish-black combustible mixture of different hydrocarbons, and is obtained in a natural state from superficial deposits in various parts of the world. The same name is applied to the pitch or residue of coal tar. The natural asphalt is chiefly used in the construction of asphalt pavements and waterproof roofing and the product derived from coal tar is used for the same purpose, and also in the manufacture of roofing paper, metal paints and varnishes, artificial fuel (briquets), etc. The grinding of asphalt gives rise to large quantities of dust, which may cause serious affections of the cornea. The fumes arising from the melting pots are liable to produce inflammatory conditions of the eyes and respiratory passages,

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and a peculiar yellowish eruption of the skin. Open pans should be avoided and Leymann recommends that the fumes should be carried into a furnace, or absorbed by a condenser charged with oil.

Brick and Stone Masons.—Ascher[35] reports that among 5695 masons belonging to the Berlin Sick Insurance Companies, there were 2181 or 38.2 per cent. cases of sickness. The mortality rate from respiratory diseases inclusive of consumption was 53 per cent., against 46.1 per cent. in the general population over 15 years of age. The mortality from diseases of the heart, digestive tract, kidneys and bladder, is also excessive. Pneumonia is most frequent in the helpers.

Hoffman's[36] Industrial Insurance statistics, based upon 1647 deaths among masons from all causes, show a mortality rate of 31.1 per cent. from respiratory diseases, inclusive of 17.7 per cent. from tuberculosis. The death rate from urinary diseases was 13 per cent.; from heart diseases 10.1 per cent. and from accidents 9 per cent. The German accident rate for journeymen was 3.8 per cent. and for apprentices 6 per cent. The Leipsic Sick Benefit records show a sickness rate of 777 days per 100 masons and a mortality hazard of 0.70 per cent.

Dust and exposure to the elements are important predisposing causes to respiratory diseases, and the sloppy character of work is conducive to rheumatic and neuralgic affections, while lime and mortar accounts for the undue frequency of eczema of the hands and wrists.

Preventive Measures.—Accidents should be prevented by proper construction of scaffold and education of apprentices. Bricks and stones should be moistened to diminish dust inhalation. Cleanly habits and the application of cold cream or vaseline will aid in the prevention of skin affections incident to cement and mortar work.

The erection of sheds, so as to provide shelter against cold or storms, facilities for warming the meals, ablutions and change of clothing is clearly indicated.

Plasterers.—Plasterers, like bricklayers and cement workers, are exposed to unfavorable working conditions, such as damp air, and, in certain stages of the work, to inhalation of large amounts of dust containing lime and plaster of Paris. If open charcoal fires (salamanders) are used for drying purposes, there is danger from coal gas. Opinions differ as to injurious character of lime dust; some authors contend that tuberculosis is less frequent among lime and cement workers than in other dusty occupations. Hoffman's Industrial Insurance statistics show that of 577 deaths among plasterers, 136 or 23.6 per cent. were caused by consumption; the degree of frequency was above the average at all ages under 65, and especially at ages between 25 to 34. The mortality from pneumonia and other respiratory diseases was 16.1 per cent., making a combined death rate of 39.7 per cent. from diseases of the respiratory organs. The Leipsic Sick Benefit records show a sickness rate of 738 days per 100 plasterers, and a mortality hazard of 0.46

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per 100. Similar conditions would probably be revealed among whitewashers if separate statistics were available.

Preventive Measures.—It is important that dust production should be reduced to a minimum, and that the general precautions recommended for masons and cement workers should be adopted.

Paperhangers.—This occupation, especially the process of scraping the walls for the removal of old paper, involves considerable dust production, and possibly also exposure to lead and arsenical poisoning if the wall paper happens to contain such toxic color pigments. A number of cases of arsenical poisoning have been recorded. (See page 4.)

There is likewise some danger if the room has been occupied by infectious patients, and disinfection has been neglected.

These risks would be more pronounced if the walls were not previously moistened, but, even when this is done, the subsequent pointing up and sandpapering of rough surfaces results in dust production. Hoffman's Industrial Insurance statistics show that of 319 deaths among paperhangers 107 or 33.5 per cent. were caused by consumption and 11.1 per cent. from other diseases of the lungs, making a combined rate of 44.6 per cent. from diseases of the respiratory organs, against a normal expected proportion of 23.6.

Preventive Measures.—Wet processes for wall scraping, use of respirators in all dusty work. Personal cleanliness and all the precautions recommended against lead and arsenical poisoning.

Emery and Corundum.—Emery and corundum wheels are employed as an abrading material, for grinding and polishing metals, stone, glass, etc., and the manufacture and use of these materials doubtless plays an important rôle in the development of pneumoconiosis. Emery is derived from emery rock, imported from Smyrna and the Island of Naxos, and is an anhydrous oxide of aluminum, containing flint, silica, and a small amount of magnetite or hematite.

Corundum is also a very hard granular variety of oxide of aluminum. The crushing and grinding of either of these materials, although carried on in inclosed machinery, involves more or less exposure to a very fine and extremely irritating dust. The wheels and stones of different sizes are made from the powdered rocks, mixed with one or more of various binding materials, such as clay, glue, shellac, India rubber, oil, sulphur, and silicate of soda. The mixture is placed in a mould and subjected to hydraulic pressure. The mixture with clay is fired in kilns and the resulting product is said to be much harder than when other binding substances, such as shellac or glue, are used; but the latter binders are more elastic and less likely to break. In order to guard against the possibility of breakage, brass wire webbing is inserted to prevent fragmentation and resulting injuries. All wheels are or should be finally tested in a metallic compartment at an exceedingly high rate of speed, over 7000 revolutions a minute, and those which pass the test without break-

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ing may be considered safe. The industry involves not only exposure to a very irritant dust, but also to lead from the babbit metal employed in fitting the wheels to spindles.

Oliver[37] states that since the introduction of the emery wheel in the Sheffield cutlery industry, the cutlers as a class have become even more unhealthy than they were in previous years. The Inspectors of the Massachusetts State Board of Health[38] report that, while the great majority of the employees in this industry appear to be well and strong, "a notable proportion present a pale and sickly appearance."

Preventive Measures.—The manufacture and use of emery or corundum wheels is doubtless dangerous to health, and should be safeguarded by reducing dust production to a minimum and adequate exhaust ventilation.

Emery and Sandpaper Making.—This occupation involves exposure to the inhalation of considerable quantities of emery powder and sand, derived from "garnet," which is a fine variety of gravel, obtained in this country in the Adriondacks. This gravel is ground, sifted, and graded, by machinery, and spread on paper covered with glue and the product is dried over steam pipes.

Preventive Measures.—Great care should be exercised to diminish dust production to a minimum, possibly by the employment of moist processes in the grinding, sifting and grading processes, for it is reported by the Inspectors of the State Board of Health of Massachusetts that in spite of the working of a 48-in. exhaust fan, to which the pipes are connected, there was a great deal of fine dust in the air. Workmen are urged to wear respirators, but it is extremely doubtful whether the inhalation of very fine dust is thus prevented.

Diamond Cutters and Polishers of Precious Stones.—This industry is to a great extent centered in a few cities of continental Europe, notably Amsterdam, Antwerp and Oldenburg. There are only about 400 men employed in the diamond-cutting shops of New York as compared with 9000 in Amsterdam. The rough diamond is usually cut by another diamond and is then embedded in a mass of molten metal, composed of about 60 parts of lead and 40 parts of tin, to which a handle is attached. The same method, although with variable proportions of the alloy, is also employed with other precious stones, and greatly facilitates the polishing process, which is done by hand, but mostly by means of rapidly rotating small iron wheels or leaden discs. In either case there is exposure to dust and lead, and the danger from lead poisoning is all the more pronounced if revolving lead discs, or a lead-containing grinding powder, are used. Some authors still maintain that during the melting of the alloy there is also exposure to lead fumes, but this is an evident misapprehension, as the volatilizing point of lead is far above the fusing point. The "setters" who prepare the diamond or gem for the cutter by soldering are, however, exposed to coal gas when charcoal fires are used to prepare the solder.

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Tracy, cited by Hoffman,[39] found 73.5 per cent. of the setters in Coster's factory at Amsterdam pale and anæmic; 57 per cent. had palpitation, giddiness, precordial distress; 56 per cent. chronic headache; 36 per cent. asthma, etc.; 90 per cent. had phthisis, and 30 of 90 men examined showed traces of lead poisoning. Oliver[40] reports that wrist-drop and colic are the prevailing forms of plumbism.

The same danger is observed in the cutting and polishing of other precious stones, which industry, in some parts of Bohemia, is not infrequently carried on in the homes of the artisans.

The undue prevalence of plumbism among the diamond cutters in the Netherlands induced the Dutch Government to offer a prize for a suitable substitute for lead in the alloy, which has not yet been awarded.

It is reported by Oliver that some of the gem polishers in Oldenburg use an alloy of tin, copper and bronze, and Rambousek, in referring to the industry in Bohemia, states that the authorities have required substitution of silicon carbide for lead discs.

The work, apart from danger of lead poisoning, involves eye strain, and is often carried on under unfavorable environments as regards light, ventilation and heating. A combination of factors, such as bad air, sedentary habits, inhalation of dust, and ingestion of lead are doubtless responsible for much of the ill health. According to Tracy, 52 per cent. of the cutters or polishers employed in Coster's factory at Amsterdam were thin and pale, 40 per cent. were asthmatic, and 33.75 per cent. suffered from headache, etc. Cases of lead poisoning are doubtless as common among the cutters as among the setters.

Preventive Measures.—Since it has been shown that an efficient substitute for lead exists, the use of lead discs should be prohibited. All polishing processes should be carried on by the wet process and guarded by hoods and efficient exhaust ventilation. Frequent ablutions and all the safeguards against lead poisoning should be invoked. Good general ventilation, adjustable work benches, and improved working conditions as regards light, air-space, and heating, are recommended.

The Glass Industry.—This industry includes the manufacture of window glass, plate glass, and all varieties of cast and rolled glass, pressed and blown glass, such as jelly cups, tableware, tumblers, goblets, lamp chimneys, lantern globes, electric light bulbs and globes, opal ware, cut glass, bottles, jars, demijohns, etc. During the last U. S. census 68,911 wage earners, inclusive of 4762 females and 3561 children under 16 years of age, were employed in this industry; 53.2 per cent. were employed in establishments where the prevailing hours of labor were 54 hours or less per week; 20.2 per cent. worked between 54 and 60 hours and 26.6 per cent. over 60 hours per week.

Glass is manufactured by the combination of a fusible alkaline silicate of potash or soda with one or more infusible silicates of lime, magnesia, iron, chromium, etc. Crown glass, for example, is composed of silica 62.8, lime

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12.5, potash 22 and alumina 2.6. Flint glass contains silica 38.2; lead oxide 48.5, potash 11.7 and alumina 2. Optical lenses and imitation precious stones are generally made of the best quality of flint glass. Most of the glasses are leadless. Arsenic is used as a de-colorizing agent, but cases of arsenical poisoning are comparatively rare, inspite of the fact that dust in glass works has been found to contain as much as 1.5 per cent. of white arsenic. Chromium and manganese compounds and various chemicals are used for coloring or decolorizing purposes. The mixing of raw materials is usually a very dusty process and the fusing and glass-blowing process involves exposure to intense light and heat. Glass-blowing by machinery is at present limited to the manufacture of glass bottles, which are moulded or blown by machines using compressed air. Plate glass is cast, and most of the table glass ware is moulded and pressed into shape by machines. It is hoped that the machine process will come into use before long in the manufacture of large glass carboys. The work of blowing large pieces of glass is often so strenuous that the pipe is passed in succession from one operator to another, which practice has been the means of spreading syphilis. A number of small factory epidemics were reported before the Internat. Med. Congress in Paris in 1876. Eysel,[41] in 1896, reported 12 instances and Brosius, in 1904, added to the number of wholesale transmissions; isolated cases are not infrequent. Hayhurst cites a case in his Ohio Survery on page 261. Cases of heat prostration are not uncommon and exposure to intense heat and light is responsible for an undue prevalence of diseases of the eye, notably chronic inflammation and dryness of the conjunctivæ, and most important of all is the large number of cataracts observed in glass-blowers. Meyhöffer[42] found this condition among 506 glass-blowers in 59 men of whom 42 had not yet attained the age of 40. Hirschberg attributes this to direct heat absorption, others suggest that the loss of bodily fluids on acount of excessive perspiration, may be a factor. It is probably caused by the combined effect of both. In a recent discussion of the subject in London, Mr. Cridland suggested the name of "ray cataract" as a more inclusive term and the President suggested the name of "furnace workers cataract" because the affection is not uncommon among puddlers (J. A. M. A., April 3, 1915, page 1186; see also page 331 of this volume). The tradition that glass-blowers are especially liable to develop emphysema of the lungs has been rudely shaken by Prettin and Liebkind,[11] who found only five cases among 230 glass-blowers who had been engaged for over 10 years in this work. Similar observations have been made in German Military bands, and we quite agree with Schmidt[11] that the elasticity of the normal lung tissue may withstand the additional strain. That the buccal cavity is taxed to an enormous extent is shown by the great dilatation of the cheeks and flabby muscular atrophy, which in older glass-blowers occasionally results in hernia of the cheeks and dilatation of Steno's duct.

As a result of exposure to excessive heat and sudden changes, catarrhal affections and diseases of the respiratory organs, which are often aggravated

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by smoke and dust in badly regulated establishments, lead the list. According to Hoffman's statistics 39.4 per cent. of all the deaths among glass-blowers were caused by respiratory diseases, inclusive of 30.1 per cent. from consumption. The German morbidity rate for respiratory diseases was 32.7 per cent. Congestive headaches from exposure to heat, and diseases of the digestive organs from excessive use of cold drinks are also quite common. Injuries and wounds, painful and inflamed callosities in the palm of the hands, from handling rough glass, and eczema, caused by glass dust, are not infrequent.

The workers engaged in dry grinding, sanding, filing, drilling, bevelling and glass polishing, especially in the crystal or so-called cut-glass industry, are exposed to the inhalation of rouge and putty powder. Oliver has found that some putty powder contained as high as 70 per cent. of lead compounds. Forty-eight cases of lead poisoning were reported in Great Britain from 1900–9 from the use of putty powder. Attempts to secure putty powder free from lead have so far failed. In France a compound of metastannic acid has been used with success and in this country most of the glass polishing and etching is done by means of hydrofluoric acid mixed occasionally with other acids such as acetic, carbolic acids, etc. This process, however, is not free from danger, as the inhalation of acid fumes is injurious, and contact with the skin is liable to cause burns in the second degree, and slow healing ulcers, with loss of finger nails, etc. Sand blasting is being employed to some extent to replace the dangerous process of etching by means of hydrofluoric acid. Dr. Hayhurst in his Ohio Survey refers to another substitute for etching, in which the design is stamped on the glass with a paste and dusted over with zinc oxide. This work is done under a hood, after which the glass is heated and the finished product resembles very closely etched ware. The different art glass processes, such as assembling in metal frames, lacquering, varnishing, etc., involve exposure to the fumes of amyl acetate, wood alcohol, benzine and turpentine, and even lead in handling and soldering metal composition frames.

Workers engaged in decorative painting and spraying of glass may be exposed to lead color pigments, and persons employed in the silvering of glass pearls are liable to develop argyria, a shiny black discoloration of the skin, caused by the absorption and excretion of silver compounds.

The danger of glass dust inhalation is plainly revealed by the undue prevalence of respiratory diseases. Hoffman's statistics show that 47.4 per cent. of the glass cutters perish from diseases of the respiratory organs, inclusive of 34.5 per cent. from tuberculosis. The presence of lead will, as in printers, naturally act as an additional predisposing factor. The average duration of life in the German glass polishers is given by Anacker as 32.6 years. Dust production is not so great in the manufacture of optical lenses, but constitutes nevertheless an injurious factor. For dangers in the manufacture of thermometers, etc., see workers in mercury, page 524.

Preventive Measures.—All raw material should be ground and mixed in

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enclosed machinery provided with exhaust ventilation. Appert's apparatus, which is operated by compressed air, should replace as far as practicable the glass-blower's pipe. Metallic and asbestos screens for protection from intense heat should be provided. Dark eye glasses as a protection from intense heat and light should be worn. All polishing processes should be safeguarded by adequate exhaust ventilation. Protection of the hands and wrists for men who handle plate glass is important. Washing and bathing facilities, special lunch rooms, change of clothing, periodical medical inspections, educational methods, and all the precautions against lead poisoning are recommended.

Mineral Wool.—The manufacture of mineral wool is closely connected with blast-furnace operations since it is obtained by subjecting furnace slag, while molten, to a strong blast such as steam under pressure. This causes a mass of fine interlaced filaments, resembling wool or cotton, which are collected in wire receptacles. The operation is naturally attended with the evolution of dust quite as irritant in its effects as glass dust. The material is also known as "mineral cotton," "silicate cotton," and "slag wool," and is used for covering boilers and steam pipes, and as a filler in ceiling spaces to deaden sound, etc. The manufacture and use of this material involves exposure to a dangerous kind of dust.

Workers in Röntgen Tubes and Röntographers.—The men engaged in the manufacture and use of Röntgen tubes are exposed, unless properly protected, to certain injurious effects of the Röntgen rays. These effects are especially liable to manifest themselves in inflammatory conditions of the skin and burns varying in degree, according to the intensity of the rays, duration of exposure, and delicacy of the skin. In some instances burns have resulted in extensive destruction of the skin and large slow healing septic ulcers. The more commonly observed effects manifest themselves in the form of a chronic inflammation of the skin, characterized by hypertrophy of the horny layer and a furrowed, dry, rough and parchment-like appearance of the skin. Fissures and painful excoriations, hard warts, deformities of the finger nails, felons and loss of hair are not uncommon. While most of the damage usually falls upon the hands of the operator, the face and other portions of the body may be involved and even cases of cataract have been observed in connection with burns of the face. Loss of hair and atrophy of the skin not infrequently gives rise to a red shiny disfiguration of the skin. In some cases, telangiectases with stellate dark spots have been observed on the back of the hands. Cases of cancer, engrafted upon chronic ulceration from X-ray burns, have been recorded. Atrophy of the testicles and sterility as a result of repeated exposure to Röntgen rays have likewise been reported. Von Jagic[45] found a marked lymphocytosis (35–52 per cent. in 10 X-ray workers and also reports three cases of lymphatic leukemia in röntographers and one in a chemist who prepared radium. Animal experimentation led him to the conclusion that both the X-ray and radium act upon the bone-marrow

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cells and stimulate the activity of lymphatic tissue. All of these effects are less commonly observed now and will continue to decrease by the employment of precautionary measures (see also page 391 and 525).

Preventive Measures.—Formerly the men engaged in the manufacture of the Röntgen tubes made the test upon their own hands and thus incurred special risks. This danger has been obviated by mounting the skeleton of the hand in a paper maché or wax model to which a leaden handle is attached. In modern establishments it is now the practice to shield the body by means of suitable sheet-lead screens, and for the protection of the eyes lead-glass observation windows and goggles are employed.

The universal use of these screens together with making the exposure as brief as practicable will not only protect the maker, but also the röntographer and thus eliminate the ill effects of the Röntgen rays, which play a very important rôle in modern methods of precision in diagnosis and also for therapeutic purposes.

Radium.—The effects of exposure to radium apparently do not differ essentially from those of the X-ray. So far, probably because of its limited employment and briefer periods of exposure, no very serious injuries have been reported. While acute forms of dermatitis, with redness, swelling, itching and burning sensations and even deep-seated ulcerations have been reported, the skin lesions are generally of a chronic type and characterized by hyperkeratoses of the lateral surfaces of the fingers.

Preventive Measures.—Sheet-lead screens, lead-glass observation windows and goggles, and careful time limits of exposure have so far afforded the best protection.

Robinson and Wilson[46] found no cases of tuberculosis among the *grinders* of *lenses* and 17 workers in an *art glass factory* of Cincinnati. No cases of tuberculosis were found among 31 persons engaged in the manufacture of *glass signs*, in which the lettering was etched in with hydrofluoric acid and sand blasting was also resorted to. Both processes were safeguarded however by an exhaust system.

Among 245 workers examined in the *stone and clay industry*, eight persons or 3.26 per cent. were found to be tuberculous; five of these were among marble workers, two in a pottery and one in a tile works. This is a much higher rate than that in most of the other industries investigated.

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CHAPTER VII

THE BUTTON, HORN, CELLULOID, AND ALLIED INDUSTRIES

BY GEORGE M. KOBER, M. D., Washington, D. C.

Button Manufacture. Vegetable Ivory. Pearl Buttons. Horn Industry. Manufacture of Celluloid Goods. Preventive Measures.

Button Manufacture.—Buttons are made of all kinds of material, such as vegetable ivory, metal, celluloid, compositions of casein, potatoes and mixtures of fossils and vegetable gums combined with pulverized carbonate of lime, feldspar, etc. In 1905 according to the U. S. census of manufacture 10,567 persons were employed in this industry; of these 5085 were engaged in making fresh water pearl buttons and 1001 in the manufacture of vegetable ivory buttons.

For lack of space, reference can be made only to these two classes and to the manufacture of horn buttons as likely to affect the health of the wage earners.

Vegetable Ivory.—The seed of the fruit of the South American ivory palm (phytelephas marcrocarpa) commonly known as the "ivory nut" is about the size of a hen's egg, and resembles in texture and color genuine ivory. In sawing the material into suitable sizes and shapes, the workmen are exposed to large amounts of coarse sawdust, which is difficult to remove by suction. The Report of the Mass. State Board of Health Inspectors for 1908 (page 85) states that one plant had spent a large amount of money in trying to adapt a blower system to its sawing machines without success. A much finer dust is produced by the machines which cut out the buttons and drill the holes, and during the polishing process, all of which can be adequately removed by suitable suction devices. The inhalation of this dust, like that of horn and genuine ivory dust, with their sharp and angular fragments, predispose to diseases of the respiratory organs, while the dust from bone is classed by Professor Roth[1] among the less offensive varieties of dust.

Since vegetable ivory is especially adapted to the application of colors, there is an element of danger if toxic pigments or dyestuffs are used.

Pearl Buttons.—In the manufacture of pearl buttons, studs and ornaments, the shells of mullosks are used; after cooking, the shells are freed from the fleshy parts, sorted into three sizes and soaked for several days in water to render them less brittle. While still wet the shells are sawed into suitable blanks; this as well as the subsequent processes such as cutting out the discs,

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boring, planing, facing, grinding, polishing and doming are all attended with the production of dust. The wage earners include a large number of females and minors. The inhalation of this dust produces dyspeptic conditions and catarrhal affections of the respiratory passages, such as chronic bronchitis which may eventuate either in fibroid phthisis or in pulmonary tuberculosis.

Of 390 deaths among the button workers in Vienna between 1895 and 1905, no less than 272 or 69.7 per cent. perished from consumption, and Teleky[2] in an examination of 150 pearl button workers in 1907 found only 93 with normal lungs.

Professor Roth states that mother-of-pearl dust is largely composed of carbonate of lime and, as such, less offensive. Reference should be made to a peculiar inflammation of the bones (osteomyelitis) which most frequently affects the bones of the forearm and hand and occasionally also the shoulder blades, jaw-bone and the bones of the lower extremities, especially of youthful workers. This disease, first described in 1869 by Englisch as attacking "pearl turners," was subsequently studied by Gusenbauer, Weiss, Fischer and others, and in all about 31 cases have been recorded. The etiology still remains obscure, but is generally attributed to the absorption of some of the constituents of the shell dust, causing embolic processes in the bone. Whatever the factors such as the grinding process, water or air may be, which predispose to this affection in Germany and France, the only two countries in which cases have been reported, the disease does not exist according to Oliver[3] among the pearl workers in the shops of Sheffield where the grinding is done by the wet process.

Neisser[4] refers to instances in a pearl factory in France of severe nose bleed, pricking in the eyes, bronchial and even gastric irritation, caused by the dust of shells from certain mollusks, the symptoms differing so radically from those produced by the genuine mother-of-pearl dust that the employees promptly stop work in these shells.

The mortality statistics of button makers in the United States from 1897 to 1906 collected by Hoffman[5] are based upon 127 deaths from all causes reported in only one of the Industrial Insurance Companies; of these 48 or 37.8 per cent. died from consumption as compared with 14.8 per cent. for males in the registration area; there were also 14 deaths or 11 per cent. from other diseases of the lungs.

Preventive Measures.—The mortality statistics from diseases of the respiratory system sufficiently indicate that the occupation as at present carried on is injurious to life and health, and that every effort should be made to reduce dust production to a minimum by wet processes and to provide for its removal by effective exhaust ventilation. This at present appears to be not wholly possible, since, "notwithstanding the best protective devices found in the way of hoods and exhausts, there was some escape of dust, as evidenced by the fact that the fine white pearl dust covered the belts, machines, walls, ceilings, clothing and often the faces of

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the workers in those establishments which provided the most improved methods of protection."[6]

The State of Massachusetts since 1910 excludes boys and girls under 18 years of age from all processes involving the evolution of dust. In the prevention of the pulmonary affections social and industrial betterment will play an important part, and as a preventive measure against inflammatory affections of the jaw Dr. Ritter, a Berlin dentist, recommends care of the teeth and astringent mouth-washes.

Horn Industry.—Horn is extensively used in the manufacture of mouthpieces for pipestems, handles for carving sets, buttons, combs, hairpins, ornaments, frames for eye-glasses, etc.

In the manufacture of horn buttons, the hoofs of cattle are generally used; after boiling to soften them they are cut by machines into pieces, while still other machines shape them into buttons; they are then stamped into the desired pattern by means of hydraulic presses; all of these manipulations are less dusty than the subsequent drilling of the holes and the polishing process.

Combs, Hairpins, Etc.—In the manufacture of combs, hairpins and similar ornaments, various materials such as horn, bone, tortoise shells, celluloid, ivory, composition material and different metals are used. Reference can be made here only to those materials involving the inhalation of dust. Apart from the liability to mechanical injuries during the process of cutting the horn into laminæ by means of circular saws, all of the manipulations such as sawing and turning of the horn, and especially the process of rounding and pointing of hairpins and subsequent rubbing and polishing, are attended with considerable production of dust. There is also an element of danger during the coloring of the goods with a mixture containing aniline dyes, red lead, lime and saleratus. Dr. Power[7] refers to the liability of horn workers to anthrax infection, but cites no specific instances. According to Hirt the inhalation of dust of animal origin is more conducive to the development of consumption than dust of vegetable origin, the consumption rates being respectively 20.8 and 13.3, and for workers in non-dusty trades only 11.1 per 1000.

Manufacture of Celluloid Goods.—The use of celluloid in the manufacture of combs, pins, frames for eye-glasses, knife and umbrella handles, billiard balls, buttons, collars, cuffs, etc., is of comparatively recent date. Celluloid, also known as fiberloid, invented by Hyatt an American in 1869, is a mixture of pyroxiline, camphor and alcohol. Pyroxiline is primarily obtained by treating vegetable fiber, usually tissue paper, with nitric acid and sulphuric acid in stone jars. This process gives rise to the evolution of nitrous fumes and sulphureted hydrogen, to which the workmen are exposed, even though the process is well guarded by hoods and exhaust ventilation. After maceration in the acid solution, the material is freed of most of the acid and washed; it is then dried and mixed with wood alcohol and camphor and the final

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mixture is kneaded and moulded or rolled into sheets of suitable sizes for manufacture of celluloid combs and other goods.

The manufacture of celluloid combs, especially the process of "shaping" and "pointing" the teeth, and the rubbing and polishing, involves more or less dust production, which can be removed to a great extent by efficient exhaust ventilation.

In recent years in order to obviate much of the hand polishing processes a dip containing glacial acetic acid and other unknown constituents has been employed, the fumes of which are, however, very irritating to the mucous membranes of the eyes and to a less extent to the nose and throat.

Fortunately, good blowers are required by fire insurance companies on account of the extremely inflammable character of the celluloid dust. There is some danger of injury to the eyes, from flying chips of celluloid caused by the circular saw employed in "shaping" the bottom of the teeth and also in the "pointing" of the teeth by sand wheels or steel burrs, unless glass fronts and efficient blowers are used.

Preventive Measures.—The chief elements of danger in the horn and celluloid industry are exposure to dust, acid and toxic fumes, especially when cyanogen compounds are used, wood alcohol, toxic color pigments, fire and explosions. The obligatory installation of exhaust ventilation is not only a fire but also a health protection; in addition some underwriters require a thermostat system of automatic sprinklers. A recent inspection of one of the Brooklyn establishments satisfied the writer that the horn and celluloid industry can be carried on under favorable hygienic conditions.

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CHAPTER VIII

THE TEXTILE AND ALLIED INDUSTRIES

BY GEORGE M. KOBER, M. D., Washington, D. C., AND WILLIAM C. HANSON, M. D., Belmont, Mass.

The Cotton Industry. Manufacture of Woolen and Worsted Goods. Manufacture of Carpets and Rugs. Manufacture of Flax and Linen. Manufacture of Lace. Manufacture of Cordage and Twine, Jute and Hemp Goods. Dye Works. Preventive Measures.

The Cotton Industry.*—The work of cotton-mill employees involves more or less constant confinement in a dusty atmosphere even in the best regulated mills. The presence of dust in the air of cotton workrooms, however, does not appear to be a prominent feature except in the first few processes which cotton undergoes after being taken from the bales. The intrinsic danger in the industry in this respect lies chiefly in the opening, picking, and carding processes, the danger varying with the construction of the mill, the amount of dirt and other impurities in the stock, the means of removing the dust, and some other factors.[1] A careful consideration of other factors than cotton dust which affect injuriously the health of the workers shows that too little attention has been given to the evil consequences of poor light (especially in certain departments), excessive heat, nauseating odors, irritating gases, the products of gas combustion, the lack of proper means of ventilation, the failure to regulate properly the introduction of artificial moisture, and want of cleanliness.

By eliminating the avoidable dangers and the associated unnecessary conditions of the cotton processes the exact danger caused by the cotton dust can be more accurately judged.

Light.—Poor light may itself reduce the physiological resistance to disease, or it may be a concomitant of a number of insanitary influences which affect the health of the worker. A detailed study by the writer of several hundred cotton mill buildings in Massachusetts showed conclusively that too little thought had been given, in mill construction, to providing for light in accordance with the kind of work to be done in a given room. Some of the finer goods mills were remarkably well constructed, however, and a few very old buildings where coarse and colored goods were manufactured were operated under commendable conditions. Many buildings had rooms of old construction, with low ceilings, small windows and small panes of glass. Some of these rooms were narrow and admitted fair light from the sides; but some were wide, and some were basement rooms which lacked both an ample supply and an even distribution of light.

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Aside from the question of mill construction, two important factors may contribute to poor light viz.: (1) neglect to keep the ceiling and walls clean and white; and (2) infrequent washing of windows, allowing them to go unwashed in some instances for several years. It is often the case that prismatic glass of different kinds and sizes is introduced into poorly lighted rooms; but unless this glass is kept reasonably clean, it is of little value. In poorly constructed and neglected rooms, with or without prismatic glass, artificial light is not uncommonly used even on bright, sunny days in the late morning or early afternoon hours; and in such rooms incandescent bulbs or gas jets are likely to be found. Even if artificial light is not used until the late afternoon hours, there is then much variation as to the time and method of lighting and the kind of light in use. In some instances the light should be turned on half an hour, or longer, before the engineer sees fit to do so; yet the employees during this time are supposed to continue their work with the same degree of accuracy and rapidity as with good light.

It is a well-established fact that either the overuse of the eyes, or the use of eyes under bad conditions, may give rise to eye fatigue or to eye strain; and many eye specialists believe that at least 80 to 90 per cent. of headaches are dependent upon eye strain.

With these facts in mind, it is impossible to ignore the probability that many individuals working by gaslight, or even electric light, in dirty, unpainted, overheated rooms, with impure air and excessive moisture, for 10 hours a day or merely for the last 2 hours during the day, use up a great deal of nervous energy, and suffer from eye fatigue, or eye strain, and its consequences.

In the weaving and spinning departments there are two distinct causes of possible injury to the employees, viz., (1) insufficient light and (2) dust.

Some employees may be exposed to both sources of injury, although it is not usual to find much dust in a weave room. The department where reasonably good light is important, in which most commonly there is considerable dust, is that of ring spinning. Here, on account of the character of the machinery and the way in which it is placed, there are very commonly dark alleys, so that the work of "finding the ends" becomes somewhat trying to the eyes, particularly in rooms that are poorly lighted.

In both weaving and spinning departments there are a number of other insanitary influences which may affect the health of the workers, the effects of any one of which are not susceptible of correct measurement. The spinning rooms are often excessively hot; the temperature in the winter months is commonly above 90°F., and occasionally above 100°F. The heat generated by the friction of the machinery in a ring-spinning room is at least sufficient to keep the room at a good spinning temperature in the winter months, provided the room is at a proper temperature on starting in the morning.

Although in the majority of the ring-spinning rooms some means of

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introducing artificial moisture is adopted, the methods of introducing the moisture vary considerably, while in some rooms no artificial moisture is introduced. In a considerable number of spinning and a large number of weave rooms the means of introducing moisture is by the old steam vapor pot, which many agents say is a detriment to a mill, both because it permits excessive steam and heat, and because the regulation of humidity is practically impossible. In addition, therefore, to poor light and some dust in many of the weave rooms and to considerable dust and poor light in many of the spinning rooms, there is commonly an excess of moisture with unnecessary heat in weave rooms, and excessive heat with frequently undue moisture in spinning rooms. Some spinning rooms have no means of artificial moisture; the air in these rooms is, as a rule, very dry.

A weave room with poor light, unnecessarily high temperature, with some dust and an excess of moisture, is not, from a sanitary point of view, a desirable room to work in; neither is a spinning room with considerable dust flying about, together with excessive heat and either undue moisture or no artificial moisture whatever. Add to these unhygienic influences two prominent factors which enter into health conditions of both weave and spinning rooms, viz.: (1) want of cleanliness and (2) lack of provision for a plentiful supply of fresh air, and an exceedingly undesirable class of rooms is represented which is conspicuously common everywhere among weaving and spinning mills. In short, the conditions commonly met with in weave and spinning rooms are: 1. Poor light. 2. Presence of carbon dioxide and carbon monoxide in the air. 3. Non-regulation or unscientific and unsatisfactory regulation of artificial moisture: (*a*) excess of moisture, undue heat; or (*b*) no artificial moisture, excessive heat. 4. Dust from the cotton and from "sizing," etc. 5. Want of cleanliness. 6. Want of provision for a plentiful supply of fresh air.

The presence of dust in the air of workrooms is a prominent feature in its influence on health. Aside from dirt and other impurities which may be in the stock, the vegetable dusts are inimical to health. Those operatives in cotton mills who are peculiarly sensitive to unhygienic influences may become seriously affected through the constant irritation of cotton dust in the upper air passages, giving rise first to dryness of the throat and later to cough and expectoration. There are many departments in which processes are conducted which expose the employees to dust, but the weaving, spinning, carding and waste rooms illustrate the important unhygienic conditions commonly associated with this disturbing element. In estimating the effects of dust upon health, the following considerations, among others, are to be borne in mind:

1. Grade of stock used, *e.g.*, quality of cotton.
2. Quantity of dust in a given room.
3. Whether the dust is constant.

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4. Type of construction of the room.
5. System of management.
6. Number and kinds of other unhygienic influences present.
7. Means of ventilation.
8. Health and susceptibility of the individual.

In some weave rooms the air is so filled with minute bits of dust as to present a hazy or "smoky" appearance throughout the room, and parts of the room are covered with the small particles which have settled thereon; while other weave rooms are practically free from such dust, there being merely a slight amount of lint or dust of microscopic fineness.

Dust in ring-spinning rooms is more constant and is greater in amount than in weave rooms. In a fine-goods mill, in which a high quality of cotton is used in a properly constructed and well-regulated room, the amount of dust present is comparatively unimportant, from the point of view of health. On the other hand, in a coarse-goods mill, where waste stock is used in a low-studded and generally neglected room, the amount, constancy and character of the dust is such as to suggest the probability of real danger.

While in both weave and ring-spinning rooms there are many unhygienic factors, the principal features of the card room are (1) the amount of dust and (2) lack of proper ventilation and effective means of removing the dust. Cotton dust in the card room varies greatly both in quality and quantity. It is always considerable, owing to the carding process of freeing and cleaning the material. In some mills it is enormous in amount, with considerable dirt as a concomitant. The quantity of dust is commonly sufficient to cause a distinct cloudiness of the atmosphere, which, in a room lacking proper ventilation, is a serious menace to the health of the operatives.

In the assorting or waste-room "sweepings" are picked over and the different grades separated. Here may be found numerous kinds of waste and dirt, including that which comes from the floors of the card and spinning rooms, and, of most importance, sputum in large quantities, which may contain the exciting causes of infective diseases of the respiratory tract.

A comparison of the important constant or possible unhygienic influences in the ring-spinning and carding departments, with emphasis upon the dust in these rooms, is as follows: *Ring-spinning*. (1) Considerable dust (constant). (2) Infectious dust (dried sputum). (3) Excessive heat (with artificial moisture). (4) Excessive heat (without artificial moisture in some rooms). (5) Want of provision for a plentiful supply of fresh air. (6) Poor light. (7) Carbon monoxide gas, and excessive amounts of carbon dioxide from respiration and combustion. *Carding*. (1) Dust (abundant and continuous where old methods of card shipping are in use). (2) Infectious dust (dried sputum). (3) Want of provision for a plentiful supply of fresh air. (4) Carbon monoxide gas, and excessive amounts of carbon dioxide from respiration and combustion.

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Card Stripping.—The introduction of the "dustless cotton card stripping apparatus,"[2] invented and placed on the market in 1911, prevents an unusual and excessive amount of cotton "fly" and fine dust (not yet removed from the cotton) from flying in the air of the carding room. In fact, the stripping device is fitted so perfectly to the card on the one end and to a dust-tight patented machine on the other that practically no dust escapes into the air of the workroom. The use of the apparatus requires a little more time than the ordinary stripping roll consumes, but the extra time is more than offset by the improved sanitary conditions in the workroom and by the better health of the employees. This card stripping apparatus is now in use in many cotton mills in Massachusetts and throughout New England and the Southern States.

Artificial Moisture.—Reference has been made to the introduction and use of artificial moisture. In England certain localities have yielded climatic conditions which have been very favorable to the manufacture of textile fabrics. In this country within recent years systems of artificial humidification have been installed in most of the mills less favorably located in order to increase the moisture in the air of the rooms. While a certain amount of moisture gives an elasticity and smoothness to the fibers which may then be carded, combed, spun and woven more easily and evenly, too much moisture is not only detrimental to many of the operations but is also injurious to the workers.

The two simplest but practically obsolete methods of introducing moisture into mill rooms are (1) the water sprinkling or "digging" method, by sprinkling water upon the floor and (2) the use of steam. The methods now generally in use are evolved from one or the other of these practices. They include the spray moistures in a large variety of patterns. The types most commonly found in Massachusetts mills are those in which a spray is projected directly into the air in the room. A discussion of the bacterial condition of the air in picker, carding, spinning and weaving departments of certain cotton mills, with tables showing average results of examination of air in these departments, is contained in a report by H. W. Clark to the Massachusetts State Board of Health in 1913 on "A Study of the Hygienic Condition of the Air in Textile Mills with Reference to the Influence of Artificial Humidification."

In spite of the modern method of humidification the writer found, from an investigation of a very large number of cotton mills, that the raising of humidity was done in a very unworkmanlike manner. Scarcely any effort was made on the part of most manufacturers to ascertain the definite conditions with respect to heat and moisture favorable to weaving and spinning, by the use of accurate thermometers and hygrometers. In the great majority of instances where hygrometers were used the instruments were untrustworthy. The writer is of the opinion that most of the men in mills, who have the care of the ordinary stationary wet and dry bulb thermometer for de-

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termining the atmospheric humidity, either neglect to keep them in order or fail to take accurate records.

A few agents or superintendents of mills have made extensive inquiries as to what degree of humidity for a given temperature constitutes the best working conditions. These men have used the self-registering hygrometer, or psychrometer, and the "sling hygrometer" with very promising results; and one of them stated that "for quickly and accurately determining the actual moisture and temperature conditions of a room, the sling hygrometer leaves little to be desired for mill use."

The diseases with which cotton mill operatives appear to be most commonly afflicted, and from the effects of which become seriously impaired in health are: pulmonary tuberculosis, pneumonia, asthma—frequently with bronchitis, influenza and rheumatism. In Massachusetts the reduction in the death rate from pulmonary tuberculosis during recent years has been much less in the four textile centers, Fall River, Lowell, New Bedford, and Lawrence, than in the State in general. Other factors, however, enter into the question as to the harmfulness of the cotton industry to health and its effects on length of life. The sanitation in all of these cities is not on the same plane. The homes and immediate surroundings of the workers are not alike. The personal characteristics, nationality, physique, educational opportunities and habits, differ; moreover, we have no accurate statistics covering the illnesses of cotton workers in connection with the particular kind of work done and the length of time spent at that kind of work. There is probably no industry today that we know less about in its effects on health and longevity than the textile industry, of which the cotton industry is a principal part. While we may reason from death returns that tuberculosis and pneumonia are the diseases most prevalent among mill operatives, does it follow that these diseases, known to be prevalent among so many different types of workers in various industries, are the direct result of mill life and work? The writer considers this one of the most important and far-reaching problems in the study and researches of occupational hygiene.

Woolen and Worsted Manufacture.*—This industry includes the manufacture of woolen and worsted goods, the production of wool felt goods and of wool hats. During the last U. S. census 175,171 wage earners were employed of whom 13 per cent. were spinners, 22.2 per cent. weavers and 64.7 per cent. in other capacities. The number of wage earners included 75,869 females and 9576 children under 16 years of age. The general processes employed in the manufacture of woolen and worsted goods are very much of the same character as in the cotton textile industry.

The most important hygienic features are connected with the sorting, carding, combing, spinning, weaving, dyeing and finishing processes.

The wool is usually sorted by females on benches with perforated tops or on tables covered with wire mesh, through which dust and dirt falls into

* By George M. Kober.

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a box beneath. This operation is of special interest, not only on account of its dusty nature, which gives rise to catarrhal affections, but also because of the danger from anthrax infection. Of 261 cases of anthrax infection tabulated by Dr. Legge 88 occurred in this industry. The disease was formerly even more common than now and was commonly spoken of as "woolsorter's disease." Those engaged in wool scouring especially in the extraction of oil by means of sulphur chloride, carbon disulphide, benzine, benzol, etc., are exposed to these industrial poisons. Sulphuric or hydrochloric acid are also used in the cleaning process.

Manufacture of Carpets and Rugs.*—The weaving of carpets and rugs involves many of the same dusty processes incident to the textile industry in general. The materials employed differ, however, more widely as wool, cotton and hemp or jute are used singly or in combination. The wool used in this industry is generally of a short and coarse fiber, and it is quite probable that the amount of dust is greater in carpet and rug mills than in other branches of the textile industry. In some establishments the raw materials are picked, carded, twisted and spun, while in other factories the yarns have been prepared elsewhere. The bales of yarn are opened and the yarn is dusted by machinery, which gives rise to considerable dust and is especially injurious if poisonous dyes have been used. The State of Massachusetts in 1900 limited the use of arsenical pigments to 0.10 grain per square yard, and aniline dyes have superseded the use of chrome dyes to a great extent. The operation of carpet weaving is not only a very dusty process but requires great care in the prevention and detection of flaws and skips. After leaving the loom the carpet or rug is carefully inspected, sheared and brushed. The shearing process is especially a dusty operation. The work of carpet weaving is hard, but the death rates compare favorably with those of other textile workers. The English statistics show that the comparative mortality was 873, which is 8 per cent. below the standard. Consumption was 22 per cent. in excess of the normal and other diseases of the respiratory system were 11 per cent. in excess. Hoffman's statistics based upon 155 deaths among carpet and rug makers, show that 23.9 per cent. were from consumption, and 16.9 per cent. from other diseases of the respiratory organs. Thompson has seen a few cases of anthrax among weavers of rugs and carpets in eastern New York.

Flax and Linen.*—Employees in flax textiles are apparently more exposed to dust and various diseases of the respiratory organs than those in woolen mills. This is especially true of the men or boys who attend the "heckling" machines.

The work, especially the spinning, is often carried on in a hot and humid atmosphere, which predisposes to diseases. According to Oliver[3] recent employees suffer from so-called "mill fever," which lasts from 2 to 3 days and is characterized by rise of temperature, malaise, nausea, vomiting and head-

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ache. This affection is attributed to the disagreeable odor given off by the oil, and the combined effects of heat and humidity.

Accidents from machinery, "flat-foot," varicose veins, and ulcers from prolonged standing, are not infrequent. Flax workers are very liable to suffer from eczema and also from a peculiar ulceration of the skin, caused by the irritant effect of lactic and butyric acids which are used in some establishments, in a solution, during the spinning of the thread. The drippings from this solution sometimes get on the barefeet of the employees and produce similar effects (Hazen). These ulcers have been repeatedly observed by Glibert in Belgium, but not by Dr. Purdon of Belfast, indicating that the process very likely differs in different countries and establishments. Dr. Purdon describes a peculiar skin eruption which affects the arms and face of the workers who remove the full bobbins from the spinning frames. The eruption resembles smallpox and is attributed to the irritant effects of flax, water and oil. Callosities on the index-fingers of "hecklers," and painful affections of the nails of the great toe in men who work barefooted, are not infrequent.

The phthisis death rate in Belfast[4] with its 30,000 persons engaged in the linen industry was 4.1 per 1000, against 1.4 for the whole of England and Wales, and 2.1 for Ireland. According to Schuler and Burkhardt 1000 linen spinners furnish annually 221.6 cases of sickness; 1000 weavers 202.7. Female operatives suffer even more, the sick rate being 249.5 and 334.4 for the respective occupations.

Lace Workers.*—An excellent description of the manufacture of lace by machinery will be found on page 746, Bulletin of Labor No. 79, 1908. This industry, which is chiefly carried on in Nottingham, consists of lace making proper, finishing, including dressing, gauffering, bleaching and dyeing. Arlidge considers the process of "gauffering" as particularly injurious, chiefly because the machines employed are heated by gas. Excessive heat and humidity are injurious factors in some of the departments.

This occupation involves little or no exposure to dust and the death rate from consumption among lace workers in Great Britain is somewhat below the average frequency in other branches of textile manufacture. But they show a marked excess in the mortality from cancer, alcoholism, diseases of the liver, nervous and circulatory diseases. The Leipsic statistics show a sickness rate of 620 days per 100 male workers and 910 for female workers, with a low mortality hazard of 0.35 per cent. and 0.54 per cent. respectively.

Manufacture of Cordage, Twine, Jute, Etc.*—This industry includes: (1) the manufacture of rope, binder twine, cordage; (2) gunny cloth and other goods in which jute is the principal material; (3) crash towelling, linen thread, etc., made chiefly from flax; and (4) the manufacture of nets and seines. During the last U. S. census 27,214 persons, including 1763 children, were

* By George M. Kober.

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engaged in this industry, of whom 51.8 per cent. were males and 43.2 per cent. females.

The primitive methods of rope making have been displaced by machine work. But it is extremely doubtful whether this has been of benefit to the operatives. The raw material arrives at the factory in bales, which on opening and shaking apart, emit considerable quantities of dust. After shaking, ordinary jute and hemp are run through softening machines, in which, according to the Massachusetts State Board of Health,[5] "they are moistened with an emulsion of oil and water, preparatory to carding. Old rope, gunny bagging and twine are run through machines which pick the fibers apart as a preliminary to other processes. Opening, picking, softening and carding are exceedingly dusty processes, and the dust is very irritating to the air passages. A fairly large proportion of the operatives show the effects of their employment, looking pale and sickly."

Apart from dust inhalation, the employment of boiling tar in the manufacture of rope and oakum, may produce tar itch. Skin rashes, caused by the oil and coloring matter employed, are not uncommon. English statistics show that the mortality from consumption among cordage makers is excessive at ages 20–34 and 55 years or over. The general death rate is also in excess of the normal mortality of occupied males. Hoffman's[6] statistics based upon 109 deaths from all causes among cordage makers, show that 25.7 per cent. were caused by consumption and 8.3 per cent. by other respiratory diseases. The statistics of the infirmary at Dundee which is the seat of the jute industry in England show an undue prevalence of lobar pneumonia among the operatives. Oliver[7] states that deafness caused by excessive noises, and impacted ear wax, is a common complaint. He also refers to the dwarfiness and anæmic condition of the Dundee employees, and justly dwells upon the baneful effects of female and child labor in this industry. The same author mentions the fact that several cases of tetanus were traced to the raw material imported from India, the organism having been recovered from the machinery dust.

Dye Works.*—Cases of poisoning by dyes are becoming less frequent in this country on account of educational methods and proscriptive regulations against the employment of arsenical coal-tar dyes. Occasional cases still occur from arsenite of sodium, which is used for fixing coal-tar dyes in Turkey red dye works, and also from orpiment dyes, which give off arseniuretted hydrogen gas and are likewise productive of eczematous affections of the skin. Much harm also results from the employment of lead salts, especially in calico printing, and from the use of chromate of lead in dyeing yarns, etc. Cazaneuve[8] has found 10 per cent. of chromate of lead in cotton yarn and as high as 18 per cent. in woolen yarn, and in the dust of rooms where the yarn was worked up 44 per cent. The women who handle and pull the yarn are literally covered with yellow dust, and quite a number

* By George M. Kober.

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suffer from the combined effects of lead and chrome. Clayton[9] has described a number of cases of lead poisoning in this industry. Among other injurious mordants are antimony and tin compounds.

Ammonia is also used in some of the dye works. Cases of poisoning in aniline black dyeing have been reported by Deardon[10] of Manchester. Neisser[11] cites the Report of a Medical Inspector in England upon the effects of aniline oil in black dyeing works, and also the effects upon the skin of chromic acid and the bichromates in these establishments. He examined 200 employees, many of whom suffered from anæmia, headache, digestive derangements, heart-burn, dizziness, palpitation of the heart, loss of will power, excessive mucous secretions and grayish discoloration of the lips, all of which symptoms were attributed to the toxic effects of aniline. Of 82 persons employed in the padding, washing and drying processes, 34 per cent. had gray lips, 20 per cent. were anæmic and 14 per cent. suffered from the effects of chrome.

Apart from exposure to industrial poisons, the work is exhausting especially in summer on account of the heat and humidity; it is also sloppy work and hence colds, catarrhal, rheumatic and neuralgic affections are not uncommon.

Preventive Measures.—The use of all poisonous dyes and mordants should be prohibited. Until this is accomplished the following safeguards recommended by the British Medical Inspector should be carried out. 1. Mechanical suctional ventilation, (*a*) at the machines where the cloth is being dyed, (*b*) at the machines where the cloth passes through the bichromate solution, and (*c*) at points where there is danger from chromate dust. 2. Protective clothing and frequent washing of the working suits. 3. Dressing-rooms and lockers for street clothing. 4. Suitable wash rooms. 5. Special lunch rooms.

The use of respirators in dusty processes and of vaseline to the skin are indicated. All dye works should have cement floors, well drained, wooden slats to stand upon during wet work, and good overhead ventilation.

An examination of 58 workers in the *cotton belt* and *rope industry* found three cases of tuberculosis and also predisposing working conditions.[12]

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CHAPTER IX

THE BOOT AND SHOE INDUSTRY

BY GEORGE M. KOBER, M. D., Washington, D. C.

Edge Trimming, Bottom Scouring and Finishing, etc. Preventive Measures.

The Boot and Shoe Industry.—During the last U. S. census 215,923 persons were employed in this industry of whom 70,449 were females and 8099 children under 16 years.

The old-time occupation of shoe and boot making has assumed a different character during the last three or four decades by the establishment of large factories, many of which are model workshops. But even now this occupation, and certainly all repair work, is still carried on in a primitive way as a home industry, and when thus conducted we may expect to find an undue prevalence of diseases of the respiratory organs, and other affections ingendered by unfavorable working conditions. Faulty position is responsible for the round shoulders, and pressure of the iron last against the chest in the operation of burnishing, etc., is the cause of a peculiar deformity of the chest, characterized by a marked depression of the breast bone and cartilaginous ends of the ribs. Callosities of the upper surface of the right thigh caused by pressure of the clamps and strap are of frequent occurrence.

Diseases of the heart, according to Sternberg,[1] are unusually common, 15.5 per cent. against an average of 10.8 per cent. in other occupations. Both he and Koelsch[2] attribute this to venous congestions caused by the stooped position and also to intemperate habits. Injuries followed by septic infections and diseases of the skin are not infrequent. Scabies appear to be unusually prevalent among the shoemakers of Vienna amounting to 38.9 per cent. against 10.8 per cent. in other occupations. Among 399 cases of tetany described by v. Frankl-Hochwart[1] 179 occurred in shoemakers. This affection is characterized by bilateral tonic spasms of the muscles of the hand and wrist and often assumes an epidemic character, which Koelsch[2] attributes to unclean habits and close sleeping quarters. Cases of plumbism caused by the pernicious habit of holding tinned tacks in the mouth have been reported. Prolonged standing may develop flat-foot and varicose veins; sedentary habits tend to induce constipation and hemorrhoids. The Leipsic statistics show a sickness rate of 642 days per 100 shoemakers and a mortality hazard of 0.68 per cent.

In the manufacture of boots and shoes in large establishments the working conditions are generally speaking more favorable. There are a number of processes, some of which are less harmful than others. The following is

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a condensed statement from the Report of the State Board of Health of Massachusetts.[3]

Cutting Department.—In this department the first process of making the shoe is begun. The men who cut the leather and lining material for the uppers with sharp knives by hand or machines, are more or less exposed to injuries. The work calls for good light.

Fitting or Stitching.—The re-enforcement of parts with cotton cloth is done by machine work, usually by boys and girls who may occupy a standing or sitting position. The same is true of cutting tongues and eyelets stays, and the perforation of tips. The work of joining the linings to the uppers is done by machines operated by women. The edges of the vamps, quarters and tips are covered by girls and women with a cement of rubber, dissolved in naphtha, to the fumes of which the operators are exposed, especially when open cans, instead of patent closed containers with automatic omitters are used. The machines for eyelets are usually operated by men, and the buttonhole machines by women. The vamps are stitched to the quarters by men and women; the latter usually work in lighter stock.

In the sole leather and stock fitting department the stock is cut out by men and channelled and the soles and uppers are ready to go to the making department, which includes, lasting, heeling and finishing and numerous subdivisions. Most of the work is performed by men and boys in a standing position.

Lasting.—In this department the upper part of the shoe and inner sole are fitted over a suitable last and the outer sole is stitched on. Among the injurious factors should be mentioned the possibility of mercurial poisoning in persons attending the sole leather stitching machines, if mercury is employed, as a lubricant, as it frequently is, in the Blake machines. Several mechanical processes connected with the lasting department also involve exposure to heat from the warming racks and the possibility of wood alcohol and benzine poisoning from the shellac and cement employed. After stitching on the outer sole the shoe is ready to receive the heel.

Bottoning and Finishing.—In the manufacture of shoe heels the workers are exposed to the inhalation of considerable quantities of leather dust and also to the fumes of wood alcohol and naphtha, from the shellac and cement which are employed. The heels are usually rough and larger than the required size, and are turned over to the heel trimmer or shaver. The heel-trimming machine requires watchful care to avoid injury or a spoiled product. Painful callosities frequently develop on the outer and upper surface of the last two joints of the left fore- and middle fingers of the operator. In "*heel scouring*" a felt wheel faced with carborundum or sand, moulded paper and cloth is used.

Edge trimming of the sole of a shoe is also done by machine work and involves exposure to considerable fine dust which should be removed by exhaust ventilation.

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Bottom Scouring and Finishing.—In this process the sole and face of the heel receive a smooth surface by means of a wheel covered with sandpaper or carborundum. In the process of Naumkeag-Buffing the sole and shank is brought into contact with a revolving wheel covered with emery cloth, or carborundum, and involves exposure to a mixture of animal, mineral and vegetable dust. The same is true of heel and breast scouring. In the final polishing process, blacking is applied, and revolving bristle brushes and cloth wheels are employed. Wax is also put on the polishing wheels. Apart from exposure to dust, the process is very liable to produce inflammatory conditions of the hands.

As a result of exposure to dust composed of leather, sand, emery, carborundum, bristles, lint, shellac, resin and wax, it is not surprising that diseases of the respiratory passages should be quite common. According to Dr. J. Beatty,[4] Medical Officer of Health of Northampton in England, the phthisis death rate for boot and shoemakers was 2.59 per 1000, against a normal average of 2.08 for the whole population. In the City of Brockton, Mass., out of 167 deaths among shoemakers 42 or 25.1 per cent. were caused by pulmonary consumption and 11.4 per cent. by other lung diseases. In Lynn in 297 deaths among shoemakers 21.9 per cent. were caused by consumption and 10.1 per cent. by other respiratory diseases. Hoffman's statistics,[5] based upon 1930 deaths among boot and shoemakers, 371 or 19.2 per cent. were caused by consumption and 14.4 per cent. by other diseases of the lungs. The excess was most pronounced at ages between 25 to 34 when out of every 100 deaths from all causes 50.3 per cent. were from consumption against a normal expected proportion of 31.3.

Three thousand and seven employees in the *boot and shoe industry* in Cincinnati were examined,[7] and only 47 cases or 1.56 per cent. of the workers presented evidence of tuberculosis. Some processes, especially *heel polishing* and *bottom sanding*, unless adequately safeguarded, undoubtedly predispose to tuberculosis. In the factories visited, powerful blowers were provided to carry off the dust thus formed.

Preventive Measures.—No state board of health has accomplished better results than that of the State of Massachusetts which, by frequent exhibits of photographs contrasting sanitary and insanitary processes, has stimulated most gratifying improvements in the working conditions of this and other industries[6] (see Hanson's Monograph). There is every reason to believe that all the health hazards connected with this industry can be removed by efficient factory sanitation, with special reference to the removal of dust and fumes.

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CHAPTER X

TAILORS, GARMENT AND LAUNDRY WORKERS

BY GEORGE M. KOBER, M. D., Washington, D. C.

Tailors and Garment Workers.—This occupation, like that of shoe and bootmakers, has also undergone marked changes as regards general working conditions, and the old-type tailor shop can be found only in small towns and villages and in connection with repair shops. It is well known that weaklings generally seek this occupation, and it is not surprising that the constrained position of the body, which interferes with free expansion of the chest, should not only favor the development of skeletal deformities, but also predispose to pulmonary diseases. While most of the work is now carried on in large establishments, the shops are frequently not much better than the homes, or tenement shops, as regards air-space, ventilation, lighting and general sanitation. According to Dr. Geo. M. Price, of 1800 shops in the cloak and suit industry examined in New York in 1911, only 3.83 per cent. had any special means of ventilation. The efforts of the joint board of sanitary control to improve the conditions and the health of the workers in this industry, in New York, have attracted the attention of the United States Public Health Service and the authorities are now conducting a special examination of the working conditions and the effect of the employees. A similar investigation was carried on by Dr. Linenthal of the Board of Health of the State of Massachusetts as early as 1908.[1] He investigated the manufacture of men's ready-made clothing, the manufacture of men's custom made clothing and the manufacture of ladies garments. The result of the inspection showed that the industry was carried on for the most part in neglected buildings. Stairs and hallways were as a rule dirty and in many instances the water-closets were foul and filthy. The working rooms were found unclean, the walls and ceilings dirty and in need of a coat of whitewash, and the windows were often so dusty as to be almost opaque. The disgusting and dangerous habit of spitting on the floor or on heaps of rags was observed in almost every establishment visited. Dr. Linenthal very justly attributes the main cause of the insanitary conditions in the clothing industry to the contract or sweat shop system. The competition among the contractors being extremely keen, their earnings often less than that of the employees, it is but natural that little or no attention is given to matters of sanitation. The shops operated directly by the manufacturers were generally in a very satisfactory condition, and efforts toward improving existing conditions in the insanitary shops of Boston have been fruitful.

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Conditions are, if anything, worse when the work is carried on in tenement homes, where one room often serves as a workroom, living room, dining room and kitchen, and long hours and child labor are the rule, in order to eke ou a mere existence. The effects of overcrowding, overwork, "speeding up" underfeeding, constrained position and exposure to dust, and carbon monoxide from ironing stoves and leaky gas tubing are plainly evinced by the pallor and anæmia of the operatives and the undue prevalence of tuberculosis and neurasthenia.

Epstein[2] states that 43.7 per cent. of the morbidity among tailors is caused by pulmonary tuberculosis, and that of 266 post-mortem examinations in 118 male and 148 female tailors, performed at the Munich Pathological Institute, 123 or 46.2 per cent. died from consumption. The records of the Milan Clinic and of the tuberculosis clinics in this country likewise show an undue prevalence of tuberculosis in garment workers. According to Schwab[3] fully 25 per cent. of 7000 garment workers in St. Louis suffered from neurasthenia. Cases of gastric catarrh, hemorrhoids, varicose veins, and ulcers and diseases of the female genital organs are also quite common and are probably caused by improper food and pelvic congestions, incident to sedentary habits, constrained positions, foot-power sewing machines, etc. Lateral curvature of the spine, occupation neuroses and visual defects are likewise frequently observed.

Cases of industrial lead poisoning from the use of silk thread, which had been weighted by a solution of sugar of lead have been reported by Eulenberg and Teleky. Cases of eczema contracted while handling silk fabrics and tarlatan dyed with arsenic containing aniline colors, picric acid, Schweinfurth green, etc., and even symptoms of systemic poisoning from arsenic, have been reported by Layet and Epstein. Hayhurst in his Ohio Survey of 1914 refers to the use of benzine in association with cleaning and cementing processes, carried on in the same quarters.

A recent investigation by the U. S. Public Health Service (see Bulletin No. 71, May, 1915), based upon a physical examination of 2107 male and 1000 female garment workers in the City of New York, shows the following: Among male garment workers, the pressers have the most robust physique. Among female garment workers no striking physical differences were observed in the various class of workers. Tuberculosis is the most prevalent disease; a postural influence may be discerned, since finishers (among males) show the greatest percentage of faulty postures.

Apart from tuberculosis, the number of cases of emphysema of the lungs, chronic bronchitis and chronic catarrhal conditions of the nasopharynx indicates that the presence of suspended matter (woolen "fly") in the air of the workshops may be a factor in the development of these diseases. Other defects and diseases were defective vision, flat feet, defective teeth and pyorrhea, nervous affections, particularly neurasthenia, defective hearing, digestive derangements and habitual constipation due to dietetic

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errors and faulty postures. Only about 2 per cent. of those examined were free from defects, but a large part of these defects are believed to be due to ignorance or neglect of personal hygiene.

The Leipsic Insurance statistics show a sickness rate of 715 days per 100 workers per annum and a mortality hazard of 0.88 per cent.; for dressmakers and seamstresses the sickness rate is 933 days, and the mortality hazard 0.50 per cent.

Preventive Measures.—The fact that during the last census year 393,439 persons, including 230,395 women and 6252 persons under 16 years of age, were engaged in the clothing industry in the United States indicates sufficiently that every effort should be made to improve the working conditions of this class of wage earners. This can be done only by absolute prohibition of all manufacturing in tenement houses, the abolition of the contract system and sweat shops, and the enactment and rigid enforcement of laws concerning factory sanitation. Even the small establishments and repair shops should be placed under sanitary control, not only to safeguard the health of the employees, but also for the prevention of communicable diseases which may be spread through the medium of infected clothing.

Laundry Workers.—We have no statistics of the number of persons actually engaged in laundry work. The last United States census shows that 124,214 persons were engaged in the steam laundry industry, of whom 81,833 were females and 675 persons under 16 years of age; about 75 per cent. of the wage earners were engaged in laundries in which the hours of employment ranged between 54 to 60 hours per week.

Those who have read "The Long Day," "The True Story of a New York Working Girl as Told by Herself," or who have critically studied the working conditions in steam laundries, realize the hardships incurred by this class of workers. The work naturally includes a number of operations. Some of the employees are engaged in opening the various bundles and marking each piece, if not already marked, and in separating the "flat" from the "starched" work. This work is regarded as quite disagreeable, as every piece has to be handled. The Medical Officer of Stockport,[4] in England, refers to the use of aniline oil in laundries as a source of poisoning to the females engaged in marking the clothes.

After being sorted, the clothes are placed in washing machines and churned for about 20 minutes in hot water with soap or washing powder. The bleaching powders are quite irritating to the skin and eczema of the hands and arms is not infrequent. The washing machines are generally run by men, and only a small proportion of the washing is done by hand in open tubs. After removal from the washing machines the clothes are placed in a centrifugal machine, commonly called the wringer, which quickly removes the excess of water, and the articles are placed on racks and rolled into the steam-heated drying chamber. From the dryer the flat work goes directly to the mangles, where it is ironed and folded; the "starched work"

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is generally machine starched, and the ironing is done partly by machine and in part by hand. The work in summer is especially exhausting on account of the combination of heat and humidity. The temperature in the mangle and ironing department is often over 100°F. but much commendable progress has been made in the alleviation of these conditions, certainly in this country, by the introduction of high studded rooms and forced ventilation. The watery vapors from the drying chambers are speedily carried away by exhaust fans, and in nearly every well-appointed laundry we find good overhead

FIG. 43.—Laundry workers. The flat-work ironing machine is provided with an excellent overhead hood connected by suction pipes with an exhaust fan. (Photo supplied by Hanson for the Mass. State Board of Health.)

ventilation and revolving fans. Cases of arsenic and gas poisoning have been reported as a result of exposure to the fumes of coal-burning ironing stoves, or defective gas connections. Accidents in American laundries are not very common, as the machines are all well guarded. According to the Medical Inspector of the State of Massachusetts,[5] in 1907 in one establishment employing 95 persons, two accidents occurred in 5 years; in another employing 54 persons two slight accidents due to carelessness had been recorded in 15 years. Sir Thomas Oliver[6] states that in 1906, 301 accidents were reported in England—46 males and 255 females. He also cites two fatal cases of tetanus which occurred in workers apparently in the same laundry: one, a girl of 15,

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ran a splinter in her finger while washing up the floor of the lunch room; the other was a man who received injuries while trying to start the engine. Oliver thinks it probable that the floor of the mess room was infected by the soiled boots of the workers.

It is rather remarkable that recent text-books make no reference to the possibility of contracting infectious diseases in the laundry business. The Massachusetts investigation[5] on this point, even among "markers" proved negative, and the general health conditions among the employees were also

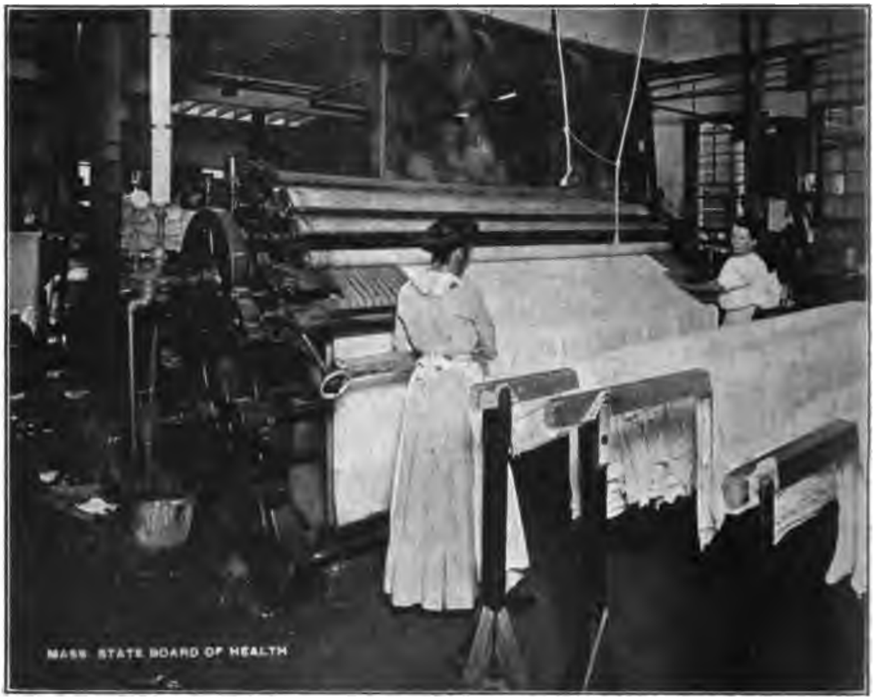

FIG. 44.—Laundry workers. The flat-work ironing machine is not protected by an exhaust system and the steam disposes through the room causing excessive heat and humidity. (Photo supplied by Hanson for the Mass. State Board of Health.)

found to be very satisfactory. Uffelmann[7] in discussing the spread of typhoid fever and cholera mentions a number of instances where the disease was conveyed to washerwomen, apparently through infected bed and body linen. It is quite possible that the danger referred to is greater in hand or home laundries.

Much of the work in laundries is carried on in a standing position; hence varicose veins, leg ulcers and flat-foot are quite common. Among other prevalent diseases should be mentioned chlorosis, diseases of the digestive organs, gastric catarrh, gastric ulcer, cardialgia, constipation, enteroptosis, retroflexion of the uterus, dysmenorrhea, rheumatism, hysteria and neu-

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ralgia, while catarrhal affections and diseases of the respiratory organs, including pulmonary tuberculosis, are also quite common. As a typical occupational disease Margoniner[8] describes a synovitis of the long extensor tendons of the right arm, beginning about the medial end of the metacarpal of the thumb and extending midway up the forearm; another favorite location of this affection is in the shoulder-joint. The women engaged in ironing and washing by hand are most frequently afflicted. Paræsthesia, or a peculiar tingling itching of the fingers, is occasionally observed in ironers. Among 165 female employees under observation for 3 years in Brooklyn, N. Y., there were 1876 cases of sickness, among them 436 cases of gastric diseases, 328 of dysmenorrhea, 124 colds, 55 of sore-throat, 36 of neuralgia and 31 of rheumatism.[9] The Leipsic statistics show a morbidity rate of 979 days per 100 ironers and washers and a mortality hazard of 0.71 per cent.

Preventive Measures.—Reasonable working hours and all the precautions already pointed out in reference to ventilation and safety devices for moving machinery, sloping concrete floors, good drainage, slatted platforms or mats, rubber aprons and boots for all sloppy work.

In examining 2648 employees in the *clothing industry* in Cincinnati, Robinson and Wilson[10] found 39 cases or 1.17 per cent. of tuberculosis. According to Hayhurst there were 228 deaths from tuberculosis among 18,793 wage earners in the clothing industry of Ohio between 1910–1912. These rates are higher than among workers in brass, copper, foundry and machine shop products. The investigators point out that a much greater percentage of the workers in the clothing industry fall within the age period, when tuberculosis is most frequent and that many weaklings seek this occupation. Low wages, "speeding up," and poor home conditions are also mentioned as predisposing factors.

Among 199 *laundry workers* four, or 2 per cent. of those examined, were suffering from tuberculosis, but not caused by the working conditions. Even the markers and listers who handle the dirty clothes consider their work free from the risk of infectious diseases. The only thing the workers fear is body lice, which they guard against. Many years ago a case of smallpox was sometimes contracted from the clothes, but never any other diseases. The older employees also testify to the improved working conditions made during the last decade, in labor-saving machinery, slatted floors, exhaust fans for the removal of steam, and reasonable working hours. In former years as one woman expressed it, "the girls were more faded looking than the clothes they were washing."

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CHAPTER XI

THE RAG, SHODDY AND PAPER INDUSTRY

BY GEORGE M. KOBER, M. D., Washington, D. C.

Shoddy. Manufacture of Wall-paper. Cardboard and Paper-box Workers.

The Rag Industry—Rag sorting is an old industry and the work is generally performed by women, whose duty is primarily to divide the rags into linen, cotton and woolen rags after which they are baled. All bales of rags are opened at the mill and the contents are placed in a beating or thrashing machine which separates the rags and shakes them thoroughly. This process is attended by the escape of more or less dust and the quantity naturally varies with the cleanliness of the stock.

The rags are conveyed from the duster or thrasher through a chute to the sorting room, where they are picked over by women who sort out all cotton, linen and silk mixtures, and remove buttons, whalebones, rubber, buckles and other foreign matter. The sorting tables have a box-like top, lined at the bottom with wire meshing through which dust and fine particles escape to receptacles below.

It has been held for a long time that the germs of infectious diseases, like smallpox, anthrax, cholera, scarlet fever, tuberculosis, diphtheria, and septic germs may cling to body and bedclothes and prove a source of danger to employees in the rag and paper industry. The danger, while perhaps overrated, is nevertheless real and would be much more evident, if federal legislation did not prevent the importation of rags without previous disinfection. Some of the states also require evidence of successful vaccination in persons engaged in the rag industry, or those employed in the manufacture of paper from foreign and domestic rags.

Shoddy is made from woolen rags. For this purpose the rags are treated in a large vat with dilute sulphuric or hydrochloric acid, which destroys whatever vegetable or cotton fiber there may be present and leaves the wool fiber intact. This process is known as *carbonizing* and may cause injury from exposure to acid fumes, unless the process is carried on in so-called carbonizing stoves which are ventilated and connected with coke condensers. Rambousek[1] considers it essential that only acids free from arsenic be employed on account of the danger from arseniureted hydrogen. The rags are next drained, spread over steam pipes and dried in a confined space, at a temperature of 100°. This involves exposure to acid fumes, which are generally guarded against before the removal of the dried rags by thorough

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ventilation and exhaust fans. The rags are next washed and run through "pickers" which reduce them to a wool of short fiber, which is mixed with new wool, spun into yarn and manufactured into cheap cloth. The grinding or picking process is extremely dusty and persons new to the work are liable to develop so-called "shoddy fever," which clinically resembles an attack of influenza. Chronic affections of the upper air passages are also quite common. During the last census this industry afforded employment to 2320 persons, of whom 480 were females.

The Paper Industry.—During the last census the paper and wood pulp industry afforded employment to 81,473 persons of whom 10,557 were females and 154 children under 16 years of age. The material from which paper is made includes rags, burlap, old paper, and wood pulp. The rags are subjected to a number of processes for the purpose of cleaning and disintegration, some of which have already been alluded to. The beating, thrashing and chopping process is carried on by machines. In the observation of about 80 establishments, the Massachusetts Board of Health found that where the cleanest grade of rags were being handled practically no dust was observed, but with the usual grade of stock, no matter what kind of duster or thrasher or chopper is used, a considerable amount of dust is evolved and in spite of exhaust fans and dust flues some dust will escape. The men engaged in the collection and baling of this dust are usually provided with respirators. The material, after passing through the chopping machines, is conveyed by an endless belt to the kettle room where it is boiled, with the addition of lime, usually in a "rag rotary," by means of steam under pressure. After thorough cooking the material is placed in the "washer;" it is then bleached by means of chloride of lime and placed in a draining vault. When sufficiently dry it goes to the "beater," with perhaps some wood pulp, where it undergoes further comminution; it is then transferred to a large tank known as the "machine chest," where it is thoroughly mixed, diluted, and stored, and from which it passes as needed to the paper-making machines.

The occupation is evidently inimical to health. Of 4857 German operatives reported by Uffelmann[2] 2437 or 50.2 per cent. were annually taken sick; the morbidity fluctuated from 9 per cent. to 112 per cent. in different establishments; there were 287 injuries; about 34 per cent. of those engaged in the handling of dry rags suffered from diseases of the respiratory organs, and only 21.9 per cent. of those engaged in other processes. Chronic bronchitis and emphysema of the lungs, impacted ear wax, and granular inflammation of the eyelids are not uncommon among the older employees. In addition to the danger from dust inhalation including also alum and resin dust, the possibility of infection with the virus of smallpox, scarlet fever, and the germs of tuberculosis, anthrax, cholera and malignant œdema cannot be ignored. There is also danger from the use of chloride of lime for bleaching purposes, from the employment of toxic colors, contining lead or arsenic, and the addition to the paper pulp of lead-containing substances. In the production of

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wood pulp by the *sulphite cellulose* process there is danger from escape of sulphur dioxide from the sulphur stoves or from the boilers.

Preventive Measures.—Greater attention should be paid to the construction and interior arrangement of establishments for the rag, paper and shoddy industry. All dusty processes should be carried on in separate rooms, partitioned off tightly from adjoining rooms where less dusty processes are carried on. Efficient exhaust ventilation is of the utmost importance for the removal of dust and fumes. The addition of poisonous color pigments and lead-containing substances to paper pulp should be prohibited. Disinfection of rags and revaccination of employees, suitable work suits including in the wet processes rubber boots and aprons, and facilities for personal cleanliness should be insisted upon.

Manufacture of Wall-paper.—In making wall-paper the paper is first passed through troughs containing colored solutions for the ground work, it then passes through machines to receive patterns and different colors. Formerly there was considerable risk from the employment of arsenite of copper and lead pigments; their use has been prohibited in some countries and replaced by vegetable or aniline colors. The employment of bronzing powders, mica and wool dust involves exposure to irritant forms of dust which must be guarded against by exhaust ventilation. The temperature in the printing department often ranges between 90° and 100°F. even in cold weather.

Cardboard and Paper-box Workers.—The Leipsic insurance statistics show that this class of workers has the highest morbidity rate among 84 occupations, viz., 1574 days of sickness per 100 workers per annum and a mortality hazard of 1.06 per cent. There is nothing in the manufacturing process which explains these hazards, unless the work is carried on as a home industry by youthful workers and without special precautions. Apart from machine accidents and dust the character of stock employed is important. If the paper happens to be colored with arsenical, lead or chrome pigments there is danger from metallic poisoning, especially in the practice of sticking on labels or loose edges, after licking them with the tongue. This pernicious habit was formerly quite common not only in paper-box factories, but in many other industries in which adhesive labels are used. As a result the so-called "stamp licker's tongue," characterized by a polished tip and small ulcers on the tongue and mouth, was quite common.

Since much of the adhesive material used is of animal origin, it is possible that the ulcers are of septic origin; at all events they offer an excellent portal of entry for septic germs and hence account for the frequent involvement of the submaxillary glands. Hayhurst[3] believes that there is danger from lead poisoning, in labelling fresh paint cans.

Much of this label licking is done by frail little girls, and the loss of saliva and its effect upon the digestive functions can be judged by the fact that, according to Miss Squire,[4] Factory Inspector of Great Britain, the little girls

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employed in labelling reels of sewing silk and skeins and balls of embroidery "moisten the adhesive labels by the mouth, sometimes to the number of 30 gross a day or more."

Preventive Measures.—It is evident that the use of mechanical contrivances and dampers to replace the dangerous process should be made obligatory.

Robinson and Wilson[5] examined 47 male and 245 female employees in four *Paper Box factories* in Cincinnati and found no cases of tuberculosis. The investigators found nothing particularly hazardous in the work except the fact that it was nearly all piecework. The dust was adequately removed by the blower system. This gratifying account presents a striking contrast to the morbidity rates of the Leipsic workers (see page 668).

Among 855 persons engaged in the manufacture of *Playing Cards* only three cases of tuberculosis or 0.35 per cent. were found. The plant was new, located in the suburbs with spacious grounds, excellent light and ventilation and general welfare work. As a result the investigators report "very little illness among the employees and this was in no way attributable to the occupation."

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CHAPTER XII

FARMERS, GARDENERS, PLANTERS AND FARM LABORERS

BY GEORGE M. KOBER, M. D., Washington, D. C.

Farmers, Gardeners, Planters and Farm Laborers.—The general death rate for 1900 in the manufacturing and mechanical industries of the United States was 13.8 per 1000 and the consumption rate 2.6, or 18.8 per cent. of the mortality from all causes.

The farming population furnished a mortality rate of 17.6 per 1000 and a consumption rate of only 1.5, or 9.5 per cent. of the mortality from all causes. Other diseases of the lungs are also less frequent. The same is true of diseases of the circulatory, urinary, digestive and nervous system; and the accident liability is also below the average. While this serves to emphasize the advantages of an out-door life, it cannot be said that this occupation is wholly free from danger. The long working hours and hardship and exposure to all kinds of weather, cannot fail to prove injurious, and as a result, colds, catarrhal affections of the eyes, nose, and throat, rheumatism, and neuralgia are by no means infrequent, especially in men who work in green houses and are exposed to abrupt changes in temperature. Much of the work, such as transplanting, grafting, weeding, and cultivation with hand tools, is performed in a stooping or kneeling position, which is liable to cause lumbago, congestive headaches, nose bleed and distortion of the femur. Of 58 cases of coxa vara observed by Bernhart[1] 49 belonged to the agricultural classes.

Loth[2] states that while on the whole the morbidity rates are more favorable, the diseases are generally more acute, the illness more severe, and the mortality rate, except from consumption, is actually above the average of most of the other occupations.

It is generally held that cancer of the skin is more common among farmers and gardeners, on account of lack of cleanliness, but Loth could not confirm this from his experience with the truck farmers of Erfurt. Among the diseases communicable from animals to men, to which farmers and stockmen are exposed, should be mentioned, foot and mouth disease, glanders, anthrax, bovine tuberculosis, and lumpy jaw or actinomycosis. Since tetanus or lock jaw is a typical soil disease, wounds and abrasions are more liable to infection in this class of workers.

Diseases of the skin caused by contact with poison ivy, sumach, and the primrose, and the affections produced by the mower mite (*leptus autumnalis*), the *pediculoides ventricosis* (or wheat bug) and the harvest bug or

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mite (*leptus irritans*) which burrow under the skin, causing itching and irritation, are occasionally met with. The mites infest especially gooseberries and hyacinth bulbs and may also cause conjunctivitis by dust infection. Other sources of ophthalmia are beards of grain, spears of grass, twigs, leaves, chaff, distribution of artificial manure, fine hair-like processes from hop leaves (hop-pickers' ophthalmia). Cases of arsenical poisoning have been reported from the use of Paris green as an insecticide.

Statistics cited by Neisser appear to indicate an undue prevalence of miscarriages in farmer's wives. Harrington calls attention to the fact that monotony of life is a potent factor in mental breakdown, as evidenced by the frequency of insanity in farmers and farmers' wives in sparcely settled regions.

Among the diseases spread by means of human fecal matter or night soil should be mentioned typhoid fever, dysentery, and other infectious intestinal diseases. We have learned only in recent years what an important rôle uncinariasis, or hook-worm infection, plays in the loss of life, impairment of health, efficiency, and earning power of the tillers of the soil in almost every part of the globe.

A correct idea of the economic aspect of this disease may be gained by reference to the chapter on Animal Parasites written by Dr. Ashford, who, in 1899, was the first to demonstrate that the hook-worm is the cause of the anæmic, dropsical, and debilitated condition, and the high death rate, of the peasant population of Porto Rico. In 1902 Professor Stiles pointed out that the same parasite is likewise crippling the South.

Mr. Rose[3] of the Rockefeller Sanitary Commission, through the courtesy of the Department of State, has been enabled to show that the hook-worm infection belts the globe, in a zone about 66° wide, extending from about parallel 36° north to about parallel 30° south, and that about 940,000,000 persons live in 46 countries where hook-worm is prevalent. It is estimated that 90 per cent. of the inhabitants of certain parts of Columbia are infected; 50 per cent. of the total population of British Guiana, with a higher percentage among the laborers on the sugar plantations. In Dutch Guiana the infection on many plantations runs as high as 90 per cent.

It is further stated that 50 per cent. of the coolie laborers on sugar and tea estates in Natal, and the same percentage of the laboring population in Egypt, are infected; that on many plantations in Ceylon the infection runs as high as 90 per cent.; and on the rubber plantations in the Malay States from 47–74 per cent.; that of the 300,000,000 people in India from 60 to 80 per cent. harbor this parasite; that the population of the southern two-thirds of the Chinese Empire is involved, the infection among the farming population running as high as 70 to 76 per cent.; and that about 70 per cent. of the entire population of American Samoa are infected.

Preventive Measures.—When we consider the vast number of persons engaged in agricultural pursuits, it is evident that greater attention should

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be paid to their hygiene. The working hours at present are excessive and should be reduced to 10 hours a day, which would offer also opportunities to vary the monotony of life. The diet is often faulty, especially the hot biscuits and greasy fried dishes, and should be improved. The clothing should be adapted to climate and seasons, and farm labor should not be done in bare feet. Personal cleanliness, frequent ablutions of soiled hands, and prompt disinfection of wounds and abrasions with tincture of iodine are indicated. More attention should be paid toward securing cheerful, airy, sunny, and healthful homes, the proper disposal of house wastes, and a pure water supply.

No farm should be without sanitary conveniences. The Department of Agriculture has published directions for the construction of suitable privies. On account of the possibility of infecting the water supply, wells and privies should not be dangerous neighbors. The undue prevalence of typhoid fever in rural districts could be materially checked by prompt disinfection of the stools of the typhoid patients. This is all the more important, since infection is often conveyed through the milk supply, and through vegetables and strawberries raised in beds which have been fertilized with infected night soil, which may thus infect consumers residing elsewhere.

Danger from Carbon Dioxide in Silos.—While cases of fatal asphyxia from carbon dioxide evolved as a result of intramolecular processes have been reported among vintners, distillers, brewers, yeast makers, in the holds of grain vessels and in peat pits, the four cases described by Hayhurst and Scott[1] are the first on record in connection with workers in silos. It is not improbable that such accidents have occurred before and death has been attributed to other causes.

These four cases of sudden death clearly indicate the necessity of greater precaution in the way of ventilation of silos, either by open doors above the level of the silage, or as suggested by Hayhurst and Scott by the use of unhinged doors, which fall in as the silage settles below them, and by open roofs. The authors state that the gas can be driven out easily by using an open umbrella, bunch of hay, or a leafy branch of a tree to promote diffusion. The extinguishing of a candle or lamp flame indicates the presence of dangerous volumes of CO_2. In case of accident oxygen inhalation and artificial respiration by means of the Meltzer apparatus should be resorted to.

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CHAPTER XIII

THE LUMBER INDUSTRY

BY GEORGE M. KOBER, M. D., Washington, D. C.

Saw and Planing Mills. Toxic Woods: Sequoia; Cocobola; Sabicu; Satin-wood; Mexican Blue Gum and Indian Rosewood; African Boxwood. Wood Preservatives, Stains and Polishes. Carpenters and Cabinet Makers. Piano and Organ Making. Chair and Rattan Factories. Whip and Walking Stick Makers. Furniture Repair Shops. Moulding and Picture-frame Makers. Preventive Measures.

The Lumber Industry.—According to the U. S. census of 1910 an average of 695,019 wage earners, inclusive of 7959 females and 5933 children under 16 years of age, were employed in this industry. The merchant saw mills afforded employment to 547,178 persons, the independent planing mills employed 112,392 wage earners, and the wooden packing box factories employed 35,449 persons. Most of the women were employed as cooks, etc.

According to the report of the Washington State Industrial Insurance Commission, 47,400 men are employed in the logging camps and lumber mills of that State alone. During the period from October 1, 1911 to September 1, 1913 there were 9661 accidents with 251 fatal injuries; 990 of the injuries resulted in permanent partial disability, and 8420 in temporary disability. Among the causes of accidents may be mentioned the following: Falling timbers, lumber, etc. Rolling and moving logs, log carriages, handling lumber, timbers, etc., flying fragments, axes, hatchets, adzes, crosscut saws, splinters, power-driven saws, planes, and wood-working machines. During the year ending October 1, 1913 there were not less than 7465 awards in this industry in that State amounting to $668,793.84.

Preventive Measures.—The accident liability is sufficiently appalling to have induced Miss Boardman on behalf of the American Red Cross to arrange for first-aid instruction in our American lumber camps. In addition to accidents the men are exposed to hard work, long working hours, inclemency of the weather and very frequently also to typhoid and hook-worm infection, vermin from avoidable insanitary conditions of camps, food supplies, kitchens, disposal of refuse, etc.

Saw and Planing Mills.—In these occupations, hard work, exposure to drafts, high temperature in the drying kiln department, the effects of incessant noise upon hearing, the inhalation of dust from planing and sandpapering machines and accidents are injurious factors.

Accident Liability.—Of 3464 accidents requiring workingmen's compensation in Austria collected by Sternberg,[1] 1074 were caused by planing machines, 283 by ripping machines, 1975 by circular saws and 132 by straight saws;

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3029 of the injuries involved the fingers, resulting often in complete loss of one or more members; 268 involved the hands and forearm, and 46 the eyes, mostly from flying fragments from the circular saws.

Wood Workers.—Apart from accidents, intense noises, from ripping and planing machines, constrained position, causing the so-called "carpenter's stoop" and exposure in certain processes to industrial poisons, the chief occupational risks are unfavorable hygienic environments, exposure to wood dust, sandpaper and sanding belts. The amount and character of dust depends upon the process in which it is evolved and the character of the wood; the dust from hard wood is finer, harder and doubtless more irritating to the respiratory passages. Sommerfeld[2] gives the mortality rate from tuberculosis among wood workers as 6.6 per 1000.

According to Roth[3] 61.7 per cent. of all the deaths among the wood turners in Berlin between 1890 and 1897 were caused by consumption; inflammation of the skin and mucous membranes are also quite common in this class of workers on account of the use of bichromate and aniline stains, wood alcohol, varnishes, etc.

Toxic Woods.—Workers in "sequoia wood," especially recent employees, are liable to suffer from catarrhal symptoms, such as sneezing, watering of the nose, smarting of the eyes, irritation in the throat, coughing, difficult breathing and oppression in the pit of the stomach.[4]

"*Cocobola wood*" is extensively used in the manufacture of tools and implements, especially for handles, and also for bowling balls. This wood evolves a very pungent and irritating dust productive of inflammation of the eyes and skin. Some persons become accustomed to its effects while others are obliged to discontinue work in that department. Similar observations have been made in Austria in connection with the manufacture of walking sticks from "cocobola" and other exotic, colored hard wood. The Factory Inspector[5] also observed an undue prevalence of diseases of the respiratory organs and insisted upon the introduction of mechanical devices for the removal of dust.

Dr. Neisser[5] refers to a tool factory at Strassburg which in 1904 furnished 15 cases of sickness out of the 20 employees, with 288 days loss of work; among these were six cases of diseases of the lungs, three of the nose and throat and one of the eyes. In this instance the authorities likewise insisted upon effective removal of the dust. Dr. A. Nestler[6] has recently studied the toxic principle of cocobola wood and believes it to be an ethereal oil, soluble in alcohol and benzol and to a less extent in water.

Sabicu wood, a product of Cuba, is said to produce a "snuffy dust" and catarrhal symptoms of the eyes and nose. The same is said of the Japanese hard wood "tagayasa" which in addition also causes dermatitis and a dark brown gunpowder-like pigmentation. The toxic action is attributed by Iwakawa to a substance closely allied to chrysarobin and like the latter agent also affects injuriously the digestive and renal functions.

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Satin Wood, Mexican Blue Gum and Indian Rosewood.—The Medical Inspector of Great Britain[5] in 1904 reports a few cases of a peculiar inflammation of the skin affecting cabinet workers engaged in handling "satin wood" panels for decorative purposes in the smoking room of a steamer. The dust from this wood (*Cloroxylon Swientenia*) affected only the unprotected portions of the body, face, neck, ears, hands and wrists. The first symptoms consisted of an intense irritation, followed by heat, redness, swelling and pain; at a later stage the skin became moist and peeled off. Some of the workmen doing the same work experienced no unpleasant symptoms. The wood from East India was found to be more irritating than the West India variety. Satin walnut appears to be harmless. Nestler[7] has recently succeeded in extracting the active principle consisting of a stearin-like substance soluble only in ether. A small quantity of it placed in contact with the skin of the forearm produced in about 5 hours a tensely swollen yellowish blister surrounded by a red area of inflammation. After the bursting of the blister an ulcer remained which required 4 weeks to heal. The dust of "cokus "wood, ebony, teak and Mexican "blue gum" is also liable to cause inflammatory skin affections. Teak wood also produces constitutional symptoms such as nausea, and vomiting. Sternberg[8] refers to "Indian rosewood" as a cause of inflammatory affections of the skin of the face, forearms and hands in the employees of one of the Vienna store fixture factories.

African Boxwood (Gonioma Kamassi).—In 1905 the Medical Inspector of Great Britain[9] reported a number of instances with toxic symptoms occurring among persons employed in the manufacture especially during the sandpapering process of weaver shuttles made from African boxwood. Investigation revealed the presence of an alkaloid in the wood which acts as a heart depressant, producing a slow and intermittent pulse, also headache, watering of the eyes and nose, feeling of sleepiness and difficulty of breathing. Some of the men were pale, weak and jaundiced and had precordial pain, nausea, cold sweats and a camphor-like odor of the breath. Oliver[10] states that Dr. Young described in 1902 symptoms such as inflammation of the eyes, dilatation of the pupils, dryness of the throat and catarrhal conditions, lasting 2 or 3 days in men making rulers from Marcaibo boxwood. Poisoning by African boxwood is subject to compensation under the British Compensation Act.

Wood Preservatives.—Persons engaged in the impregnation of railroad ties, shingles, etc., with tar or creosote are liable to suffer from vesicular and other skin eruptions not necessarily confined to the hands of the worker.

Wood Stains and Polishes.—Reference has been made on page 674 to the use of bichromates for wood stains, and on page 546 to the toxic effects of red aniline used for staining purposes in imitation of mahogony, or to impart to this wood a richer color; similar instances are doubtless quite frequent. In a recent invetigation at Budapest it was found that the prolonged working with stains containing wood alcohol may cause optic neuritis,

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simply by the evaporation of the spirit and several workmen in a cabinet factory were suffering from incipient atrophy of the optic nerve. As a result of Dr. Daifis'[11] investigation, which also revealed the presence of wood alcohol in a few of the most expensive liquors, monthly inspections of the workmen have been ordered, and it is expected that more stringent legislation will be enacted.

The so-called "polishers' itch" an eczematous condition of the hands and arms is quite common among wood polishers. The causative agent being either petroleum, paraffine oil, shellac dissolved in wood alcohol or in alcohol denatured with pyridine bases or wood alcohol. The use of varnish may give rise to occasional cases of plumbism, and Sternberg[12] reports that old topers not infrequently drink the shellac mixture leading to the formation of shellac concretions in the stomach.

Carpenters and Cabinet Makers.—According to Sternberg[13] of 10,071 male workers in Vienna during the year 1904, 5341 were taken sick; of these 1506 or 28.1 per cent. suffered from tuberculosis and other diseases of the respiratory passages. The death rate from tuberculosis in the registration area in the U. S. for 1909 was 10.1 per cent. Apart from dust inhalation this class of workers are liable to suffer from callosities and contracted tendons of the palm of the hand from the use of the chisel and those using hand planes involving rotary movements of the wrist and forearm not infrequently develop occupational neuroses and peculiar inflammation of the tendons of the dorsal surface of the thumb. Workers in parquetry and scrapers of floors are very liable to develop inflammation of the bursa in front of the kneecap, with accumulation of fluid therein (housemaid's knee). Faulty positions and unsuitable work benches naturally favor the development of spinal curvature and other deformities. Continuous standing predisposes to varicose veins, ulcers and "flat-foot," affecting especially the left side. (See also wood stains and polishes.)

Piano and Organ Making.—These industries include a great number of separate processes; of hygienic interest are those connected with dust production, the inhalation of bronze and exposure to wood alcohol in the varnishing and polishing department. Cases of lead poisoning have been reported among solderers, varnishers and bronzers of piano frames and also in men, who work on pipe organs. Other hazards are acid dipping and electroplating, brazing and welding.

Chair and Rattan Factories.—The splitting and sorting of rattan is a very dusty process and extremely irritating to the respiratory passages. There is more or less danger from exposure to the fumes of sulphur, quick lime and chlorine employed for bleaching the rattan, and likewise from hydrofluoric acid, which is used not only for bleaching purposes, but also for the extraction of its silicates. (See also wood stains and polishes.)

Whip and walking stick makers employ a variety of material such as rattan, wood, rawhide, glue, cotton thread, aniline dyes, varnishes, etc.

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There is always considerable production of dust in the splitting and turning of rattan and wood. Other injurious factors are bad working conditions, the employment of chrome and other toxic stains, which together with impure varnishes and polishing materials not infrequently cause inflammatory and even suppurative inflammation of the hands. Neisser[14] also reports cases of suppurative skin affections in persons employed in the curving of walking-stick handles, which had been treated with a solution of acetate of iron, although the older employees appeared to be exempt from such effects.

Furniture Repair Shops.—The work is generally carried on in insanitary shops, and the persons engaged in renovating old furniture and upholstery are exposed to the inhalation of dust of a mixed character possibly containing disease germs. (See also Upholstery, page 577.)

Miscellaneous Wood Workers.—In the *tub* and *pail* industry the employees, many of whom are youthful wage earners, are exposed to the inhalation of fine wood and sandpaper dust. In the cutting, trimming, turning and polishing of *shoe lasts* large amounts of dust are evolved and inhaled.

Moulding and Picture-frame Makers.—In this branch of industry, the various processes are dusty and often carried on without adequate ventilation, while in the bronzing and gilding department there is danger from the inhalation of bronze, wood alcohol and amyl acetate and so-called "banana oil." Leadless paints are almost exclusively used for picture mouldings.

Coopers, like wood turners, have a high mortality rate from consumption and diseases of the respiratory system, in which the inhalation of hard wood dust probably plays an important rôle. There is also exposure to metal dust, blacksmithing, sand blasting and painting. The men engaged in shellacing the interior of casks are exposed to the toxic effects of the wood alcohol. (See Breweries, page 694.)

Preventive Measures.—Since dust inhalation and accidents constitute the most important risks, it is evident that all machines should be properly guarded and mechanical means be provided for the removal of dust; for the latter purpose, hoppers, suction pipes and blowers have proved quite efficient especially in connection with turning and sanding machines. Some of the workmen claim that the exhaust flues cause disagreeable drafts and rheumatism of the finger joints, but this is a minor objection and could be prevented by wearing suitable gloves. It is also evident that persons handling toxic woods, and stains and polishes should protect their hands and resort to frequent ablutions. All processes should be carried on in well-ventilated rooms, and the vessels containing wood alcohol should be kept closed until used.

Robinson and Wilson[15] examined 230 persons employed in the *carriage* and *wagon industry* in Cincinnati and found only one case of tuberculosis. This is attributable to 79.6 per cent. of the workers being over 25 years of age and 42.6 per cent. over 35. The blowers attached to the dust producing machines (belt-sanding) removed a large part of the dust; steady work and fair wages also played a rôle.

But a small percentage of metal grinding is done in carriage factories. Varnishing

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usually has an injurious effect upon health, however, yet no sickness due to the process was found.

Among 270 employees engaged in the *manufacture* of *furniture*, only three cases or 1.1 per cent. were tuberculous. Much of the work, *i.e.* sawing, planing, turning and belt-sanding is dusty, but powerful blowers eliminate this hazard to a great extent; 58.1 per cent. of the employees were over 35 years of age. The sanitary condition (toilets, wash rooms, and general cleanliness) was fair in three and dirty in one of the factories.

Among 60 *moulding* and *picture frame workers* no cases of tuberculosis were found, although four were classed as much below the normal standard of health. Some of the men engaged in bronzing were pale and complained of headaches. The fumes of amyl acetate were very noticeable in spite of the employment of powerful exhausts.

Among 664 employees (94 females) engaged in the manufacture of *pianos*, seven cases of tuberculosis, or slightly more than 1 per cent. of those examined, were found. A powerful blower system carried away all dust produced by the machines. The factories were provided with good toilet facilities, wash room, sanitary drinking fountains, lunch rooms, etc. Two of the active cases of tuberculosis were employed in the varnishing department and exposed to the fumes of naphtha and dust produced in sand papering, but both were also addicted to the use of alcohol. The reporters do not consider the occupation hazardous as regards tuberculosis.

Among 273 workers engaged in *cooperage* and the manufacture of *bungs* and *cigar boxes*, seven cases or 2.56 per cent. of those examined were tuberculous. The blowers attached to dust producing machines were not always successful in removing all of the dust. The investigators point out other unfavorable conditions, such as temperature, humidity, alcoholism, dissipation, bad housing and family histories, as possible predisposing factors. Only one case, a cooper, seemed to be due more to occupation and bad working conditions, than to any other one thing, although even in this case dissipation was also present.

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CHAPTER XIV

OCCUPATIONS INVOLVING EXPOSURE TO THE INHALATION OF ORGANIC GASES, ETC.

BY GEORGE M. KOBER, M. D., Washington, D. C.

Flaying and Bone Yards. Rendering Plants. Manufacture of Tallow. Soap Industry. Glue Makers. Basic or Thomas Slag. Artificial Manure. Preventive Measures.

Occupations Involving the Inhalation of Organic Gases, Etc.—Whether the effluvia from sewers, stables, stock yards, rendering establishments, glue, candle and soap factories, hide depots, fertilizer plants, etc., are injurious to health remains an open question. Some authors insist that the olfactory organs are alone offended and point to statistics which indicate that the duration of life of such employees is above the average, quite forgetful of the fact that weaklings rarely engage in such occupations. It is reasonable to assume that the effluvia, consisting as they do of ammonia, hydrogen and ammonium sulphide, are fully as injurious as sewer air, which, judging from animal experimentation, appears to increase the susceptibility to infectious diseases by diminishing the power of resistance.

In privy pits and sewers the danger is intensified by an excess of carbonic acid and corresponding deficiency of oxygen in the air. Special precautions should be taken to exhaust the foul air before sewer employees or scavengers are allowed to descend. The general effects of foul odors upon those unaccustomed to do work in the so-called "offensive trades" are nausea, vomiting, headache, loss of appetite, diarrhea, general depression, and weakness. (See also Sulphureted Hydrogen in the list of Industrial Poisons.)

Flaying and Bone Yards.—Every community provides for the collection and disposal of dead animals, which is usually done by contract. The animals are taken to some establishment beyond the residential limits, flayed and worked up, so as to utilize the skin, hair, fats, bones, horns, etc. After the animal has been skinned and the mane, tail, hoofs, and horns removed, the carcasses are cooked in a boiler for the recovery of fat and bones and the fleshy residue is used for the manufacture of artificial manure. Apart from exposure to extremely offensive odors there is more or less danger of the transmission of infectious diseases communicable from animals to man, such as anthrax, glanders, tetanus, tuberculosis, and hence all such work should be done under strict sanitary control. Moreover, during the fly season there is distinct danger from septic infection, by biting flies, in all occupations involving the handling of decomposing animal matter, and every effort should be made to abate the fly nuisance and protect the workers by suitable screens.

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Rendering Plants.—In some establishments the refuse from dead animals and garbage, such as fat, meat trimmings, bones, etc., is treated with benzine, carbon disulphide, or by heat for the extraction of fats. The products are oils, grease and tankage. When heated in tanks the fat separates and is drawn off into barrels and sold for the manufacture of soap, wagon grease, etc. The tank residue is subjected to pressure and the meat scraps are converted into feed for poultry, or utilized for the manufacture of artificial manure. The bones are either cleaned and dried and sold for the manufacture of buttons, etc., or are ground up and converted into fertilizers. The tanks are usually provided with ventilating pipes which carry the odors and gases to the furnaces, where they pass through the fire and are destroyed. In spite of this provision there is much reason for assuming that the evolution of volatile fatty acids and acrolein are responsible for the undue prevalence of catarrhal affections of the eyes, air passages and of the stomach.

The Manufacture of Tallow.—Tallow is a mixture of olein, stearin and palmitin, chiefly the first two, and melts at a temperature of between 100° and 120°F. The better grades are obtained by rendering beef or mutton fats, but inferior qualities used for soaps are secured from the refuse fats already referred to, and even from whale or fish oil. Dr. C. F. W. Doehring[1] has described in detail the methods employed in the manufacture of tallow: "There are a series of cylinders of 1200 to 1500 gal. capacity employed in rendering the fats when the tissues are not required in a condition otherwise than is suitable for manure. The cylinders are filled above a false bottom with the crude fat and steam is admitted by a foot valve and perforated pipe at 50 or even 100 lb. pressure. . . . . After 10 to 15 hours the steam is cut off and the cock and safety valves are opened, and after settling, the layer of tallow is drawn off through a series of cocks. The cover of a discharge hole in the bottom of the cylinder is then raised by a rod, and the residue falls into the tub beneath. To remove the last traces of tissue it is necessary for the tallow to be washed, melted and strained. This process extracts more tallow from the fat than any other. Sometimes the fat is rendered at the atmospheric pressure by boiling a mixture of the fat with one-fourth of its bulk of water, containing 2–3 per cent. of sulphuric acid."

Several processes are employed for bleaching and purifying tallow. Apart from subsidence, mechanical filtration and aeration, various chemicals have been used, such as potassium carbonate, or potassium permanganate, or bleaching powder in the proportion of 1 per cent. or potassium chlorate in the proportion of 0.03 per cent. with the addition of hydrochloric and sulphuric acid. In some establishments, the tallow is purified by heating with soda and salt solution and boiling the unsaponified portion with a 2½ to 3 per cent. solution of alum. It is very evident that the workers are exposed not only to very offensive odors but also to a number of toxic fumes.

The Soap Industry.—During the last census about 13,000 persons were

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employed in the United States in soap making. The manufacture of soap and candles is usually carried on in the same establishment, and bcause of the offensive odors evolved the occupation has been considered unwholesome. The inspectors of the State Board of Health of Massachusetts do not share this opinion and report that the employees as a class appear to be in good health. The Leipsic statistics for female tallow and soap workers show 1085 days of sickness per annum per 100 members and a mortality hazard of 1.14 per cent.

In the manufacture of soap, animal fats or oils, such as olive, cocoanut or palm, containing the fatty substances olein, palmitin and stearin, are heated with either caustic potash or soda. This converts the fatty substances into oleic, palmitic and stearic acids, which combine with the alkaline base and form oleate, palmitate and stearate of potassium or sodium, while glycerine is set free and saved as a by-product. Hard soap results when soda is used as a base, and soft soap is the product when potash is employed.

In addition to the materials mentioned various coloring matters are employed. For example, the marbled or mottled appearance of castile soap is produced by the addition of ferruginous matter. Toilet soaps are frequently colored with harmless aniline dyestuffs and scented with essential oils and artificial perfumes which may cause headache. The most dangerous agent employed is nitrobenzol, which not infrequently produces malaise, headache, giddiness, nausea and other symptoms of nitrobenzol intoxication. Dr. Grün[2] suggests that some of the chronic affections of the kidney and bladder may be caused by the perfumes and chemical dyestuffs employed. The manufacture of medicated soaps containing sulphur, tar, etc., like toilet soaps, involves more complicated processes.

Apart from exposure to volatile fatty acids, acrolein vapors and soap dusts, the employees may sustain injuries from machinery, burns from caustic alkalies, and eczema from exposure to acids. Wounds and abrasions are not infrequently followed by erysipeloid infections. It is generally believed that exposure to the vapors of fatty acids is productive of catarrhal affections of the upper air passages, and Grün accounts for the undue prevalence of acute and chronic gastric catarrh among soap makers by the fact that they swallow a sufficient amount of alkaline vapors to neutralize the hydrochloric acid of the gastric juice, and that fatty acids ingested also act as chemical irritants. All of these hazards can be reduced to a minimum by hoods and exhaust ventilation.

Glue Makers.—Glue is usually an impure gelatine derived from boiling animal substances such as fleshings, scraps of hide, hoofs, horns, etc. The material is first washed and then soaked for a few days in lime water in suitable tanks for the purpose of loosening the hair. This is removed and dried by steam heat and sold for plastering purposes. The residue is washed for the removal of lime and exposed to the action of steam in suitable tubs. The liquid glue is drawn off from below and allowed to solidify in pans to a

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hard jelly, which is then cut into slices, transferred to screens and thoroughly dried. Glue is generally sold in thin, hard and brittle cakes, although sometimes it is ground up in the factory. This involves more or less dust production. The residue in the tubs is further treated for the extraction of fatty matter.

The glue manufactured from by-products of the fish-preserving industry is usually evaporated to the required consistency and sold in a liquid form. In the manufacture of so-called Cologne glue, which is a pale strong glue, considerable chloride of lime is used for bleaching purposes.

The manufacture of gelatine for dietetic purposes is carried on along the same lines, except that none but pure and wholesome stock is used, and the entire process is conducted with special regard for cleanliness.

Preventive Measures.—While it is true that the workers in these so-called offensive trades generally appear to be in good health, it should be remembered that, as a rule, only men of rugged physique engage in these disagreeable occupations. As already indicated the inhalation of organic gases and of volatile fatty acids and acrolein is not free from danger and should be guarded against by copious ventilation and exhaust flues. Personal cleanliness is likewise important and all wounds and abrasions should be treated with antiseptics.

Basic or Thomas Slag.—In the manufacture of steel by the Gilchrist-Thomas method a slag is formed, which contains from 10–25 per cent. of soluble phosphoric acid and naturally led to its utilization as a fertilizing agent. This involves crushing and grinding operations of a very dusty character, and hence diseases of the respiratory organs are quite common. In some of the workers ulcerations of the mucous membranes have been observed and the undue prevalence of pneumonia among this class may reasonably be attributed to the fact that the pathological changes, produced by the corrosive action of quicklime present in the dust, favor infection with the pneumococcus.

Foreign statistics show an undue morbidity rate especially from pneumonia, conjunctivitis, and eczema. In recent years a process of granulating basic slag, under the influence of a stream of water, has been introduced, which, while it obviates the dangers of dust production, is attended with the evolution of sulphuretted hydrogen. This should be guarded against by copious ventilation. It should be stated that granulated slag, apart from being used as a fertilizer, is also utilized in the manufacture of cement, artificial stone, etc.

Artificial manure is also made from various materials rich in phosphate of lime. For this purpose phosphorites, cuprolites, bone dust, guano, etc., are employed. The grinding of the raw material involves exposure to caustic dust and should be carried on automatically in enclosed ball mills. The material is then mixed with sulphuric acid which renders the insoluble phosphates soluble. During this process, hydrofluoric, silico-fluoric, hydro-

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chloric and carbonic acid vapors are evolved and also sulphur dioxide and sulphuretted hydrogen. These gases should be promptly removed by acid proof exhaust fans and condensed. Cases of poisoning from nitrous fumes may occur in the manufacture of fertilizers in mixing Chili nitre with strong acid superphosphates. When ground bones are used, the fats are first extracted by means of benzine, which contributes an additional element of danger.

My former student, Dr. C. F. W. Doehring,[1] has described the conditions observed in some of the fertilizer plants, and emphasized the danger from biting flies, the clouds of dust and exposure to acid fumes in some of the superphosphate factories.

In addition to the materials already referred to, the following are sources for artificial manure. The deposits of native guano consist of the excrement and carcasses of sea fowl, but many of these deposits are now exhausted and artificial guano is made from the tank refuse of dead animals, garbage, carcasses of whale and fish, after extraction of the fats. Other sources for artificial manure are seaweed, soot, damaged oil cakes, nitrate of soda, sulphate of ammonia, the latter chiefly a by-product of gas works, also dried blood, refuse of hoof, horns, leather and wool.

In examining 581 employers in different *soap factories* in Cincinnati, Robinson and Wilson[3] found 10 cases or 1.7 per cent. of tuberculosis. The manufacture of soap powder is attended with the production of much dust containing soda ash, which in addition to the blower system frequently compels the employment of respirators. Apart from this, long working hours, "speeding up" and low wages are referred to as deleterious influences.

Among the 66 employees of a *fertilizer plant*, only 1 case of tuberculosis was found, and he was a colored coal passer, who had 12 hours of work a day and sometimes slept in the fire room. The low incidence is attributed by the investigators to the fact that the majority of the employees belonged to a selected class and 71.2 per cent. were over 35 years of age, and hence beyond the most dangerous age period for tubreculosis.

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CHAPTER XV

THE INDUSTRY OF FOOD STUFFS, ETC.

BY GEORGE M. KOBER, M. D., Washington, D. C.

Grain and Milling Industry. Flour Milling. Bakers, Confectioners and Pastry Cooks. Manufacture of Glucose and Starch. The Sugar Industry. The Fish Industry. The Slaughtering and Meat Packing Industry. The Manufacture of Oleomargarine. Lard Making. Dairy Industry. The Canning and Preserving Industry. Breweries, Distilleries, etc. Manufacture of Vinegar. Manufacture of Carbonated Waters. Artificial Ice. Cold Storage. Chloride of Sodium. The Tobacco Industry. Tobacco Testers. Tea Testing. Hotel, Restaurant, and Saloon-Keepers. Domestic and Personal Service. Barbers and Hair Dressers.

The Grain and Milling Industry.—*Grain Threshing.*—It is well known that even the modern threshing process has not materially diminished the liability to dust inhalation. Fortunately, the exposure to this mixture of dust of an organic and inorganic origin is usually for a short duration, but even as it is, we not infrequently observe acute bronchial catarrh and inflammatory conditions of the eyes, nose and throat and quite commonly also intense itching of the skin.

During the season of 1914 numerous fires occurred in the State of Washington in connection with the threshing operations, especially in the threshing separator. These fires were of an explosive character and numerous persons were seriously injured, usually by fire, and in a few cases, by the force of the explosion.

Investigation rendered it reasonably certain that the cause of the fire was an unusually large amount of stinking smut (tilletia tritici) in the wheat which contains from 4–5 per cent. of inflammable oil. An exceedingly dry season increased the amount of organic dust from broken grain and straw, and also increased the combustibility of both smut and dust, and the amount of static electricity.[1]

While the source of ignition has not been positively proved, it would appear that static electric sparks, which are generally present in the cylinders of the separator, are the cause in the majority of instances, and hence it is urged that the cylinders of the separator be grounded by means of an electric brush connected to the ground by wire, attached to an iron peg driven a foot or two into the ground.

Workers in Grain Elevators.—The amount of dust is very much less in handling grain after it leaves the threshing machines. More or less dust, is, however, gathered in transport and is also produced by the attrition of the individual grains during shipment. The dust from rye and wheat grain on account of the sharp particles of the husk is more irritating than that caused

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by oats, but the latter give off a larger volume. The dust from corn usually contains large quantities of mineral matter and fragments of husks. The construction and equipment of modern grain elevators reduce exposure to dust to a minimum; nevertheless the men engaged in shovelling the grain toward the mouth of the elevators and those who work in the bins, turning over corn or grain to prevent heating, are exposed to clouds of dust. Most of this work is done by casual labor and the injurious effects would be more pronounced if the employment were constant instead of seasonal. No extended statistical material is available to determine conclusively the unhealthful character of this occupation. Hoffman[2] found that of 24 deaths from all causes among grain handlers and elevator men 5, or 20.8 per cent. died from consumption, as compared with 14.8 per cent. in the general male population in 1906.

Preventive Measures.—While much has been done to reduce the dangers from dust inhalation by automatic processes, no effort should be spared to render the devices for the arrest and removal of dust more effective. In the meantime the workers so exposed should protect their nostrils and mouths by handkerchiefs as the ordinary respirators soon become clogged and embarrass respiration.

Flour Milling.—In spite of the tremendous growth of commercial mills, there are still a large number of custom mills, where the facilities for the arrest and removal of dust are not nearly so good as in the large patent rolling process establishments. But even in modern mills where the cleaning, grinding and bolting is done in inclosed machinery, there is always more or less exposure to grain and flour dust, while the men engaged in dressing the buhrstones which are still in use in some of the custom mills are also exposed to mineral and metallic dust. If statistics were available it would probably be found that these men, and those connected with the cleaning of the grain preliminary to the milling, furnish the highest percentage from diseases of the respiratory system; the flour and bran packers, counters, sweepers and warehouse men would probably come next in frequency. The inhalation of flour dust, in spite of the smooth and less irritating character of the individual particles comprising it cannot fail to prove injurious to the respiratory organs. Indeed there is much reason for assuming that the lodgment of flour dust in the minute ramification of the bronchial tubes and air vesicles apart from clogging up the air passages and acting as an irritant, may result in collapse of a small area of the lung (atelectasis) or in a dilatation of the vesicles and an abnormal collection of air in the lung tissue, technically called emphysema, and popularly known as "miller's asthma." Other diseases of the lungs are even more common. According to Hirt, 20.3 per cent. of all the diseases affecting millers are pneumonia; 9.3 per cent. bronchial catarrh; 10.9 per cent. consumption; and 1.9 per cent. emphysema a total of 42.4 per cent. from diseases of the respiratory passages. These rates are about 11 per cent. in excess of the statistics furnished by Hoffman[2] who found

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that of 256 deaths in millers, in this country, 31.6 per cent. were caused by diseases of the lungs, inclusive of 40 deaths, or 15.6 per cent., from consumption and 29 deaths or 11.4 per cent. from pneumonia. The statistics of Hirt cover an older period and the difference is largely due to the less harmful processes employed in modern mills. But the New York State Bureau of Labor reports for 1906 a very high death rate from pneumonia in millers.

Cases of chronic indigestion are not uncommon among millers, which the writer feels disposed to attribute to the swallowing of flour dust. Since uncooked starch is not readily digested, it is very liable to undergo fermentation and thus injure the gastric mucosa. Millers occasionally suffer from inflammatory conditions of the skin, impacted ear wax and partial deafness. Other sources of danger in connection with flour milling are exposure to sulphurous acid fumes when used for bleaching purposes, accidents from moving belts and machinery, fire and explosions.

According to the preliminary report of the miller's Committee of Buffalo[3] in 13 explosions investigated in recent years in the milling industry in this country, 78 men were killed and 119 injured and the total damage to the property exceeded $2,000,000. Dr. H. H. Brown's laboratory studies have shown that grain dusts, especially from oats and yellow corn, are even more inflammable than standard coal dust and develop higher pressure on explosion. Apart from the generally recognized source of ignition such as the use of open lights or naked flames, the committee mentions electric sparks from motors, fuses, switches and lighting systems, static electricity produced by friction of pulleys and belts, and the introduction of foreign material in grinding machines.

The Committee found the latter in recent years a very frequent cause and expresses the hope that some system may be devised by which the foreign material might be removed before it reaches the mill.

Preventive Measures.—Since cork dust explosions are often caused by particles of iron producing sparks in passing through the mill, a like cause may be surmised in this instance, and Doehring's recommendation that the cork be passed through a magnetic separator before being placed in the comminuting machine would be equally applicable for the prevention of explosions in the flour industry. The recommendations of the Committee may be summarized as follows: Complete lighting systems, portable electric lamps instead of lanterns and naked lights, the inclosing of the electric light bulbs in strong wire guards; the possible use of vapor proof globes, and the locating of all fuses, switches, starting boxes, motors, etc., at points where no dust is present. The receiving bins from the grinding machines to be as small as compatible with the operations, as increased size gives increased space for dust clouds and therefore opportunities for more violent and destructive explosions. Above all else dust should be prevented from escaping into the air of the workrooms.

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Bakers, Confectioners and Pastry Cooks.—The preceding remarks concerning the inhalation of flour dust apply equally to this class of workers, except that there is a less intensive exposure and that millers are also liable to the inhalation of grain dust. At all events Hirt's German statistics are somewhat in favor of bakers, whose mortality rate from diseases of the lungs was 28.2 per cent. against 42.4 per cent. in millers; their consumption rate was 7 per cent. against 10.9 per cent. in millers, and 11.6 per cent. in confectioners and the pneumonia rate was 8.4 per cent. against 20.3 per cent. in millers. Hoffman's[4] statistics based upon 1357 deaths among bakers in the United States, from all causes, are not so favorable as they show a death rate of 34 per cent. from diseass of the lungs and air passages, inclusive of a consumption rate of 20.4 per cent., while the confectioners reveal a mortality rate of 39 per cent. from diseases of the lungs inclusive of a consumption rate of 22.2 and a pneumonia rate of 13 per cent. The excessive American rates may be due to more unfavorable environments of our smaller bakeries, for it is very doubtful whether in any other country, except perhaps in London so many bakeshops are located in basements, wholly unfit for the purpose.

The excessive rates in confectioners may be due to several factors: first, because it is quite common for delicate individuals to engage in this work; second, certain operations, for example the dipping of chocolate candies, involve a temperature between 66° and 68°, which in summer is secured by refrigerating pipes and the exclusion of the outer air, with corresponding pollution of the air of the workshops; lastly, confectioners either inhale or ingest more or less pulverized sugar. It is known that lodgment of this dust upon the teeth and gums, unless removed by frequent cleaning, favors fermentation processes and decay of the teeth, and there is much reason for assuming that such a condition may likewise favor the proliferation and virulence of disease germs, such as the pneumococcus, which is quite frequently found in the buccal cavity of normal individuals.

Other factors prejudicial to the health of bakers are exposure to heat, coal gas, impure air, long hours and hard work; this is especially true of city bakers whose mortality is very much higher than in towns and villages. Apart from diseases of the respiratory passages, anæmia, probably caused by chronic gas poisoning, acute infectious diseases, digestive disorders, and diseases of the heart are quite common.

Bakers, and all others engaged in handling dough, are liable to suffer from eczema of the hands and arms ("bakers itch") and also evince a peculiar susceptibility to scabies, pimples and boils. Conjunctivitis is likewise quite common and youthful employees frequently develop "in-knee" "knock-knee" and "flat-foot."

Fractures, burns and scalds were always common. Other accidents, such as contusions and incised wounds, have increased with the introduction of modern mixing, kneading and cutting machines.

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Preventive Measures.—While Health Boards have been quite active in their efforts to secure better sanitation of bakeries, it is evident that more should be done in the way of legal requirements. There is no good reason why bakeshops located partly below ground should be tolerated, and why the manufacture of the "staff of life" should not be carried on under more favorable sanitary environments, as regards air-space, light, ventilation, general and personal cleanliness, with benefit to the producer and consumer alike. It is to be hoped that machine labor will displace at least the "drudgery" of the bakeshop and that day work with reasonable hours will take the place of night work. Italy passed a law in 1908 for the abolition of night work in bakeries.

Manufacture of Glucose and Starch.—During the last census year this industry afforded employment to 5827 persons of whom 4773 were wage earners. Starch is made chiefly from corn, although wheat and potatoes are also used. There is no statistical data upon which to base conclusions as to the injurious health factors in this industry. It is evident, however, that the work around the steeping tanks is connected with exposure to dampness and inhalation of putrefactive gases, especially in the "alkaline process," in which the corn is steeped in water at a temperature of 70°–140°, from 3–10 days. The handling of the corn or wheat, preliminary to maceration, involves exposure to grain dust; while the drying of the starch, which is done in rooms with a temperature between 125° to 170°, involves exposure to starch dust and abrupt changes in temperature, all of which cannot fail to affect adversely the health of those exposed. As in the case of flour mills there is also danger from explosion of dry starch.

Glucose is made commercially by treating starch with diluted sulphuric acid. The resulting solid product is called grape sugar, and the syrup, glucose. In this process the workmen, in addition to the other injurious factors, are also exposed to sulphuric acid fumes, and arsenical poisoning of the acid is made from arsenical pyrites.

Preventive Measures.—Provisions should be made for the removal of dust and obnoxious gases. The employees should not be obliged to remain in overheated rooms any longer than is necessary; and actual work in storing and removal of the starch from the drying departments should not be done until the temperature is below 90°.

The Sugar industry practically covers three industries, viz., the manufacture of "beet sugar," "sugar and molasses," and "sugar refining." The total number of persons employed in the United States in 1909 was 24,047 14,047, of whom 34.9 per cent. were engaged in the beet sugar factories, 22.1 per cent. in the cane mills and 43 per cent. in the refineries.

The work, like the manufacture of starch, involves exposure to dampness, excessive heat, humidity and steam, especially during the condensation process where the atmosphere of the room is generally above 95° and the relative humidity about 96°, with corresponding dangers incident to abrupt changes

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in temperature. The employees not infrequently work stripped to the waist, and, as a result of moisture and contact with the sticky substances, develop eczematous conditions of the skin. Cases of inflammation of the lymphatic vessels, caused, according to M. Gaillot by the staphylococcus pyogenes aureus found in the residue of molasses, are occasionally observed. In his opinion this organism may produce virulent toxins and fatty acids by the conversion of saccharine material into lactic, acetic and butyric acids, and the soluble poisons, as well as the microörganisms, may enter through the hair follicles. A number of the processes also involve exposure to industrial poisons, such as ammonia, sulphurous fumes, carbonic acid, sulphuretted hydrogen, and the inhalation of bone black dust. There is also a slight liability to phosphorus poisoning from the bone black refuse of sugar mills. The regeneration of animal charcoal involves exposure to offensive gases, including the products of putrefaction and fermentation. In the beet sugar industry, especially when the diffusion method is employed, an explosive mixture probably containing carburetted hydrogen, has proved a source of danger to the operatives. The refuse waters from such plants have been regarded for some time as a source of stream pollution and are therefore subjected to treatment before discharge into open water courses. The men employed in connection with the sedimentation tanks are exposed to the inhalation of sulphuretted hydrogen and other impure gases. The Leipsic statistics show a morbidity rate of 1443 days per annum per 100 workers in sugar refineries.

Preventive Measures.—Adequate provisions should be made for ventilation and general cleanliness. Prof. Roth[5] points out that only strong men with an active skin should be employed. Ample washing facilities for the prevention of eczema should be available. The water-closets should be so located as not to involve exposure to abrupt changes in temperature. It is gratifying that, according to Neisser,[6] these recommendations are being complied with in a number of plants.

Fish Industry.—The occupation of fishermen necessarily involves exposure to the inclemency of the weather, but it cannot be said that the occupation is dangerous to health, since it occupies the eighth rank in the comparative mortality of 22 occupations tabulated in England and Wales.

The work of fish curing has some special interest because of the employment of a large number of females, and also because during the herring season it involves long and irregular working hours and even night labor. Miss Mary M. Patterson[7] in discussing the herring, cod and haddock curing industry with special reference to female labor, points out that as a result of long hours and exposure, rheumatism, bronchitis and diseases of the lungs are not uncommon. Severe cuts, slow healing and septic wounds are not infrequent. In speaking of injurious environments she observes, "the very ground they stand on is often a menace to health, for, in the absence of proper paving and drainage, the earth becomes impregnated with decaying

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organic matter, which 'smells to heaven' and creates a constantly foul atmosphere, not completely counteracted by the open-air surroundings."

The hygiene of this industry has been very much improved since the establishment of modern canneries, and, while the primitive conditions still prevail in the curing of herring and cod fish in different sections, the handling of most of the sea food is now carried on under more favorable working conditions.

Preventive Measures.—Special attention should be given to the construction of the buildings; cement floors, wooden lattice work for the employees to stand on, suitable tables so that the water will drain away from the workers, rubber aprons and boots, good ventilation for the removal of bad air or steam and suitable dressing and lunch rooms should be provided. Much of the work is now done by machines, and the method for the collection and utilization of the offal have been perfected, so as not to constitute a menace to public or private health.

The Slaughtering and Meat-packing Industry.—The average number of persons engaged in this industry during the last census year was 108,716; of whom 7413 were females. The total number of animals slaughtered for food was 88,358,815; hogs represented 60.2 per cent., sheep and lamb 16.7 per cent.; beeves 15.4 per cent.; calves 7.4 per cent.; and goats and kids $\frac{3}{10}$ of 1 per cent. This tabulation does not include the retail trade. The hygiene of this industry has been materially improved in modern establishments, but is still quite unsatisfactory in the rural districts and wherever primitive methods are in vogue. The employment involves hard work, exposure to dampness, and a liability to infection from diseases communicable from animals to man, such as anthrax, bovine tuberculosis, glanders, tetanus, actinomycosis (lumpy jaw), and septic conditions of cuts and abrasions. Butchers who are in the habit of eating raw meats may become infected with tapeworm and trichina.

According to Leiser,[8] of 4200 male butchers in Berlin, during the 3 years from 1902–1905, an average of 2125, or about 50 per cent., came to the attention of the Sick Insurance companies. Of these 38.6 per cent. were treated for injuries and scalds incident to the trade; 19.2 per cent. for sexual diseases; 7.2 per cent. for diseases of the respiratory organs; 5.4 per cent. for rheumatism; 4.7 per cent. for skin diseases, chiefly eczema (the last two groups are largely influenced by exposure to wet and drafts); 4.6 per cent. for diseases of the digestive system; and 3.7 per cent. for "flat-foot" and varicose ulcers, probably caused by standing too long on their feet. The excessive rates from sexual diseases may be due to more frequent exposure, especially as animal food stimulates the sexual appetite and butchers are proverbially well fed.

Preventive Measures.—It is clearly the duty of the State to exercise a strict sanitary supervision of this important food industry, for the protection of the producer and consumer alike, and this supervision should extend to the smaller establishments in the country, and the numerous meat and

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sausage shops in the city. Wherever unsatisfactory conditions are found, they should be removed and brought up to the standard which prevails in clean, decent, and properly managed establishments.

Manufacture of Oleomargarine.—The manufacture of oleomargarine originated in 1868 with Mege Mouries, a chemist, who at the instigation of the French Government undertook a series of experiments for the purpose of securing a substitute for butter, at less cost, which might be used by the army and navy and the wage earners of France. The original process of Mouries was patented in the United States in 1873.

The product consists of oleo oil, neutral lard, butter, cream, milk and salt. Refined cotton-seed oil is sometimes used in limited quantities in the cheapest grades. Oleo oil is made from the choicest fat of the beef. It is taken out of the animal, washed in several changes of water and placed in a vat of ice water to stand until the next day. It is then cut up and transferred to large steam-jacketed kettles in which it is boiled under constant stirring. It is next piped to kettles on the floor below, where it is clarified, after which it is conducted into tanks on the floor below and allowed to stand until the stearin separates. It is then placed in linen cloths and the oil is extracted in a hydraulic press and barreled. The residue in the cloths after pressing, commercially known as stearin, is utilized for the manufacture of candles.

Neutral lard is obtained from the leaf lard of the pig and after thorough washing is placed in cold storage for 24 hours; it is then cut up into shreds and cooked and after steaming presents a snowy white appearance. The oleo oil and neutral lard in the proportion of about 27 per cent. of the former and 35 per cent. of the latter, are churned with cream or milk, salted, run through cold water, worked in a butter worker, placed in suitable packages, and labelled according to the U. S. Laws, "Oleomargarine." The product is subject to an internal revenue tax of 1 ct. per pound, and if colored to resemble butter it is subject to a tax of 10 cts. a pound.

Lard Making.—Another important industry carried on in connection with slaughtering and packing houses is the production of lard, of which there are several kinds. In rendering leaf lard, part of the fat separates at a temperature between 105° to 120°F., and constitutes, after washing with a trace of sodium carbonate, common salt or dilute acid, the choicest and richest part of lard known as neutral lard—referred to in the preceding paragraph. Leaf lard is obtained by steam heat under pressure from the residue, after the neutral lard has been piped off. "Choice" kettle lard is derived from the remaining portions of the leaf and from the backs of the hogs. Other grades of lard are derived from the head, the fat of the small intestine and other viscera, trimmings and other fatty parts and is sold under the euphonious names of "prime steam" or "refined" lard.

These occupations are of importance as the process of rendering animal fats is attended with the evolution of fatty acids, such as palmitic, stearic, oleic, margarine acids, and at higher temperatures necessary for the melting

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of tallow, the so-called acrolein vapors. Under certain conditions acetic and butyric acids, and also ammonium sulphide, sulphuretted hydrogen, pyridin, picolin and lutiosin may be evolved. Acrolein produces itching in the throat, irritation of the eyes, lachrymation, conjunctivitis, irritation of the upper air passages and bronchial catarrh. Dr. Grün[9] believes that ingestion of fatty acids is liable to produce gastric catarrh, and that many of the diseases of the stomach in this class of workers are primarily caused by excessive consumption of scraps of fat meat fished out of the rendering kettles.

Injuries from moving machinery, burns and infected wounds are not uncommon, and erysipelas occurs sufficiently often to justify reference by some of the German authors to such terms as "margarine" and "tallow erysipelas" and "zoomotic erysipeloid," meaning thereby that the infection is conveyed through the raw products. The wound infection is characterized by inflammation of the lymphatics and glands, and a moderate temperature. It can scarcely be regarded as a typical streptococcus infection, but nevertheless suggests prompt antiseptic treatment of all wounds and abrasions.

Preventive Measures.—Copious exhaust ventilation for the removal of offensive odors and gases.

Dairy Industry.—The exact number of employees in this industry as a whole cannot be stated. During the last census year there were 19,380 persons engaged in butter factories; 7164 in cheese factories, and 4962 in condensed milk factories. The average number of female wage earners was 1420, of whom 987 were in the condensed milk factories.

The hours at dairy farms if not long are unseasonable and involve more or less night work. The milkers suffer in rare instances from spasm of the flexors of both hands and fingers. Instances of infection from cowpox, tuberculosis, from handling diseased udders or infected milk or cream, ringworm from leaning their face against the flank of an affected animal, and occasional cases of infection with the germs of foot and mouth disease and actinomycosis, or lumpy jaw, have been reported.

The employees in dairies, especially those engaged in the pasteurizing and bottling departments, are exposed to a damp and steamy atmosphere and the work under foot is sloppy. The strong borax and other alkaline solutions used in the cleaning of bottles, utensils, etc., and also employed to renovate rancid butter, quite frequently produce eczematous conditions of the hands and arms, unless guarded against by the employment of machinery and rubber gloves. The hygiene of persons employed in the manufacture of butter, cheese, or condensed milk does not materially differ.

Preventive Measures.—Fortunately education, legislation and competition have done much to reduce the dangers to a minimum. Nevertheless watchful care is still needed to prevent the transmission of disease through the medium of milk and dairy products. This is true not only of the diseases already mentioned, but also of typhoid fever, diphtheria and

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septic sore-throat, in which infection takes place from handling of the milk by bacillus carriers, or by washing the utensils in infected water. In our present state of knowledge nothing short of pasteurization will confer immunity from so-called milk-borne diseases.

The Canning and Preserving Industry.—The average number of persons engaged in this industry during the last census year[10] was 71,792 of whom 59,968 were wage earners; of these over one-half were females, and 4246 or 7.1 per cent. were children under 16 years of age. The census covers four classes of establishments, viz.: (1) Plants whose chief products are canned and preserved fruits and vegetables, including dried and packed fruits put up in packing houses; (2) establishments whose chief products are canned and cured fish, including pickled, smoked and dried fish; (3) establishments whose chief products are canned osyters and clams; (4) establishments whose chief products are pickles, preserves, jellies, sauces, etc.

A large number of the establishments are located in rural districts. The working hours are irregular and often very long. The work itself is sloppy and involves exposure to dampness, and in the cooking and packing department a steamy atmosphere prevails, which upon condensation helps to saturate the clothing of the workers. The surroundings are often extremely insanitary, especially in the jam and jelly industry, and in the rural districts the temporary camps are a distinct menace to the health of the people. The work of lifting and carrying buckets or baskets weighing from 40 to 60 lb., from the supply court to the peeling room and from there to the canning department, is not an easy task for women and children, who receive but 4 cts. for skinning a 40-lb. basket of tomatoes and a similar wage for peeling apples and fruit, averaging about 6.3 cts. per hour. The amount of work and exposure can be readily judged when it is remembered that during the rushing season some of the employees have weeks of 72 and even 81 working hours.[11]

As a result of long hours and exposure, catarrhal affections of the respiratory passages, rheumatism, and anæmic conditions are by no means infrequent, and sore hands and cut fingers are not uncommon sights. The soldering process involves exposure to lead and acid fumes. The men employed in the preservation of evaporated fruit are also exposed to the fumes of sulphurous acid; this agent is also employed for the fumigation of hops.

Preventive Measures.—The recommendations referred to in the fish industry are equally applicable to canning establishments. There is no reason why mechanical conveyors and machine labor should not supersede much of the hand labor. Health authorities should insist that hoods and exhaust drafts for steam and fume processes are provided and that the equipment and sanitary arrangements observed in some of the establishments are made compulsory for all others, in which the conditions are unsatisfactory. Special attention should be paid to the sanitation of the working camps in rural districts.

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Breweries, Distilleries Etc.—A number of operations connected with this industry involve hard work and exposure to extremes of heat and humidity. This is especially true of the malting and brewing processes. The work of turning the malt, performed in a temperature between 158–176° is laborious and should be displaced by automatic machinery. The men engaged in the brewing department are exposed to the combined effects of heat and humidity, and those employed in the cooling rooms to a temperature between 38° and 40°; hence rheumatic affections are quite frequent, amounting to 20–22 per cent., against 15–16.5 per cent. in other occupations. Work connected with the fumigation of hops, vats and casks involves exposure to sulphurous acid fumes, and the employment of ""salufer" as a preservative and antiseptic may prove injurious as it contains about 2 per cent. of silicofluoric acid. (See also Arsenic, page 12.) The men employed in the fermentation vaults are exposed to large volumes of carbonic acid, unless special care is taken for the liberation of these gases. This is likewise true of wine cellars, champagne factories, and carbonated water bottling establishments. Another source of danger in breweries and distilleries is the employment of wood alcohol as a solvent for the shellac to coat the interior of large storage casks and vats. Quite a number of fatal cases from this cause have been reported in recent years in New York, Buffalo and Chicago.

The death rate of brewers, distillers and rectifiers in this country during the census year of 1900, was 19.7 per 1000, which is far above the average of other occupations. Tuberculosis and other diseases of the lungs, heart, kidneys, nervous system and of the digestive organs lead the list. This we find also to be the case in Great Britain and Germany. The number of deaths from tuberculosis and other diseases of the lungs among brewers in Great Britain was 518 per 1000, against an average of 416 in the general population. Sommerfeld calculates that 52.4 per cent. of all the deaths in brewers are caused by pulmonary diseases, inclusive of 47.2 per cent. from consumption. According to Weihrauch[12] 36.4 per cent. of all the deaths among Munich brewers were caused by tuberculosis, 19.7 per cent. by heart disease, 11.1 per cent. by accidents, 8.02 per cent. by diseases of the kidneys, 7.4 per cent. by tumors, etc., 4.9 per cent. by pneumonia and 3.1 per cent. by typhoid fever. The average duration of life is about 32.6 years (Koelsch).[13]

The Leipsic statistics show a morbidity rate for brewers and malsters of 1106 days per annum per 100 workers and for workers in yeast factories 863 days.

The undue prevalence of rheumatism, diseases of the lungs, heart and kidneys may in part be due to exposure incident to the work, but the intemperate habits doubtless play even a more important rôle in their causation. (See also the alcohol habit.)

Preventive Measures.—In all fermentation processes cases of asphyxia may occur, on account of an excess of carbon dioxide and deficiency of oxygen. Such instances have been reported in breweries, distilleries, yeast

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factories and bottling establishments. Hence copious ventilation should be provided near the floor level for the removal of CO_2. There is also danger from carbon monoxide and arsenical fumes if the products of combustion are allowed to pass into the malt or other drying chambers.

Modern hot air kilns and automatic malt-turning machines should be provided. Special precautions are necessary in the sulphuring process of malt and hops, so that no one enters until after thorough ventilation of the premises, the efficiency of which can be tested by holding a moistened strip of blue litmus paper into a partly opened door, this will turn red, if sulphurous acid fumes are still present.

Artificial Ice.—The manufacture of artificial ice by the ammonia and brine process is quite generally carried on in connection with breweries and cold storage plants. The work is wet and sloppy and involves exposure to very low temperature (0–50°F.) with abrupt changes. In most of the modern plants the ice is handled and stored by machinery, but injuries are by no means infrequent, and cases of poisoning from ammonia fumes are not uncommon.

Cold Storage.—A temperature of between 32° and 40°F. combined with excessive humidity is inimical to health. In all cold storage plants, where ammonia is used for refrigeration purposes, special precautions are necessary to prevent leakage and also to provide helmets or an oxygen breathing apparatus, suction pumps, etc., for the safety of the workers in case of an accident.

Vinegar.—In the manufacture of vinegar there is frequently escape of acetic acid fumes, alcohol, aldehyde, acetic acid ether and other low oxidation products of alcohol. These may not only prove injurious to the health of the workers but constitute an economic loss, which can be prevented by the employment of an hermetically closed automatic apparatus.

Manufacture of Carbonated Waters.—This occupation, apart from exposure to large quantities of carbon dioxide, involves considerable risk of injuries from bursting bottles, as a result of gaseous pressure during the bottling, wiring or capping process. The injuries from flying glass fragments may involve the loss of an eye, or serious wounds of the blood-vessels. The work is often carried on under unfavorable environment, and involves standing in the wet and working in wet clothes, with its attending consequences.

Preventive Measures.—Copious ventilation, cement floors properly drained; wooden slat floors, wherever wet processes are carried on; waterproof clothing and rubber boots; and, last but not least, suitable wire face guards and gauntlets.

Spices.—The grinding and packing of mustard, pepper, allspice, nutmeg, vanilla, etc., involves exposure to irritant dust owing to the presence of essential oils and alkaloids, which may give rise to catarrhal conditions of the eyes and upper air passages, and also in some instances, notably in the

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manipulation of "vanillon," an impure specie of the vanilla bean, to irritation and inflammatory conditions of the skin.

Chloride of Sodium.—The manufacture of chloride of sodium or table salt involves exposure to a moist steamy atmosphere, during the evaporating process, which is productive of catarrhal conditions of the upper air passages. The effects of exposure to chlorine are evinced by the fact that Müller[14] in examining 165 salt workers, mostly packers and grinders, found 45 cases of nasal catarrh, 45 perforations of the nasal septum and 9 recent ulcers.

A peculiar form of dermatitis characterized by the appearance of acne, redness, and œdema of the face, eyelids, and tips of the ear is frequently observed among workers in the drying process. This, together with catarrhal symptoms of the upper air passages, loss of appetite, and digestive derangements, is attributed to the irritant vapors of hypochlorites and should be guarded against by hoods and copious exhaust ventilation.

The Tobacco Industry.—The cultivation of tobacco and its subsequent storage in well-ventilated barns for the purpose of "curing" may be regarded as purely agricultural pursuits. The stripping of the leaves from the stalks, the sorting, drying and packing and unpacking involves more or less inhalation of dust and the pungent odors of tabacco. The work in the "tobacco dryers" involves occasional exposure to excessive heat and the inhalation of fine tobacco dust and a certain amount of risks from dust explosions due to the greasy and inflammable character of the dust. But on the whole none of these preliminary processes, according to the Austrian statistics, appear to be as detrimental to the health of the wage earners as the manufacture of the final product. This is probably accounted for by the fact that the preliminary processes are seasonal in character and the exposure of a comparatively short duration.

Manufacture of Tobacco, Cigars, Cigarettes, Etc.—This industry in 1905 afforded employment in the United States to 159,408 wage earners of whom 57,174 or 42.2 per cent. were females and 5274 or 3.9 per cent. were children under 16 years of age; 135,318 or 85 per cent. of the workers were engaged in the manufacture of cigars and cigarettes; 23,044 in the manufacture of chewing and smoking tobacco; and 946 in the manufacture of snuffs. Quite a number of authors state that the manufacture of snuff involves the greatest amount of fine dust production but this is an evident error as the tobacco for snuff is ground up in a moist state. The manipulation of tobacco in a dry state results, however, in more or less dust production; this is especially the case in the unpacking, the separation and sorting of leaves and subsequent cutting process and also in the manufacture of cigars. It is a source of satisfaction that the use of machines has to a considerable extent displaced hand labor in a number of the processes. It is estimated by the Bureau of Census, that fully 85 per cent. of the cigars and nearly all of the cigarettes are now manufactured in this country by machinery, a similar revolution has taken place in the manufacture of smoking and chewing tobacco, where granulating,

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shredding and packing machines perform the work formerly done by hand. There is no doubt that these modern methods, quite apart from the hygienic aspect of doing away with the objectionable and nasty practice of finishing cigars with the aid of saliva, will result in a distinct improvement in the health of the employees. On the other hand, it must not be forgotten that the preliminary processes and even the use of machinery unless provided with dust-removal devices will still involve exposure to dust containing more or less nicotine. This, together with exposure to volatile, pungent tobacco odors incident to the various processes of selection, blending, fermentation, flavoring and saucing of tobacco, cannot fail to prove injurious.

Heucke[15] examined the dust in different establishments and found that it contained 0.56 per cent. of nicotine; Stephani[15] calculates that a cigar maker in turning out between 5–600 cigars, each weighing 10 grams, manipulates in close proximity to his mouth and nose every day about 5 kg. of tobacco, containing over 100 grams of nicotine and adds that when it is considered that the 1/100,000 part of this amount subcutaneously injected is liable to produce serious symptoms of nicotine poisoning, even a minimal percentage in the dust may suffice to produce specific toxic effects. In proof of this he presents Kostial's observations, showing that 72 of 100 of the female employees in a Vienna tobacco factory suffered during the first 6 months from congestive headache, palpitation of the heart, precardiac anxiety, weakened heart action, intermittent pulse, pain in the stomach, heart-burn, vomiting, diarrhea, loss of sleep and appetite, neuroses, general fatigue and loss of strength—a clinical picture which corresponds to that observed in animal experimentation with nicotine.

Much has been said and written concerning the health injurious factors in this occupation and opinions differ. Some authors doubtless overestimate the risks; others deny all dangers; indeed some authors maintain that tobacco dust exerts a protective influence against infective organisms, and instance the fact that during the cholera epidemic of Hamburg in 1892 there was only 8 cases among the 5000 resident cigar makers. Be this as it may the statistics of the U. S. Census Bureau show that among 23 occupations tabulated, cigar makers and tobacco workers occupy the second rank in the mortality from tuberculosis, the first place being held by marble and stone cutters.

Hoffman's[16] Industrial Insurance statistics based upon 141 deaths from all causes show a mortality of 34.8 per cent. from consumption and 9.9 per cent. from other diseases of the lungs, which is far in excess of the rates for bakers, confectioners and others exposed to the inhalation of vegetable dust. The Austrian statistics cited by Professor Roth[17] and based upon the morbidity and mortality statistics of 35,000 tobacco workers show that among the causes of deaths, tuberculosis occupied the first place, other diseases of the lungs came next, followed by diseases of the circulatory and digestive organs.

As evidence of the specific irritant properties of tobacco dust Professor

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Roth[17] refers to the frequency of inflammatory affections of the skin, eyes, throat and of dental defects. Brauer was the first to point out that the sharp angular fragments of tobacco dust are very liable to produce lesions of the mucous membranes. Among the surgical diseases Stephani[18] mentions felon and cellulitis; he also points out a very low accident rate in tobacco workers. Neuralgia is not uncommon and a peculiar form of neuritis affecting the right forearm has been described by Kostial.[18] Galezowski[19] found cases of amblyopia in tobacco workers who did not use tobacco in any form. De Schweinitz[20] reports the case of a young woman suffering from loss of vision whose symptoms disappeared when she left the tobacco factory. Dowling[21] reports a case of tobacco amblyopia in a woman who had worked in the factory for 5 or 6 years, but who had never used tobacco. Such instances are doubtless quite rare. Dr. de Schweinitz writes me that the woman's work involved immersion of her arms for several hours a day in a rather strong solution of tobacco. Diseases peculiar to women were twice as frequent than among workers in the textile industry; chlorosis and anæmia are also frequently observed.

According to Rosenfeld and Picraccini, female tobacco workers are more liable to miscarry. While this confirms the conclusions reached by Dr. Roger S. Tracy of New York as early as 1873, it is at variance with the results of an investigation made at Giessen, a German tobacco town, in 1906, and hence more extended investigations are called for. The latter inquiry revealed, however, a greater frequency of post-partum hemorrhages and a tendency to profuse menstruation among this class of workers. The statistics presented by Stephani[18] covering some tobacco towns in Baden from 1889–1901 also indicate an undue prevalence of tuberculosis. In 1893 the German government promulgated regulations covering the installation and operation of tobacco factories and provided a minimum air-space of 7 cubic meters which was increased in 1907 to 10 cubic meters (353.16 cu. ft.) for each worker. Rules concerning ventilation, the removal of dust and the exclusion under certain conditions of female and child labor were also enacted.

The undue prevalence of consumption is in part explained by the fact that many weaklings enter upon this work; while this must be conceded, as well as the fact that in the absence of adjustable seats and work benches the constrained position of the workers favors a predisposition to pulmonary diseases, yet the same is true of youthful confectioners and candy makers, and, although likewise exposed to the inhalation of organic dust, these latter show a much lower death rate from diseases of the lungs. All of this tends to the conclusion that we are dealing not only with the inhalation of irritant vegetable dust, but also with a toxic agent, viz., nicotine, the combined effects of which probably account for the lower general resisting power to disease.

Tobacco Testers.—Men who are in the habit of sampling different brands

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of tobacco by smoking and chewing have been known to suffer from the effects of nicotine poisoning, and like tea tasters have been obliged to limit their work or give it up all together for the relief of nervous symptoms and visual defects.

Tea Testing.—The testing of tea is done by experts in the tea business. For this purpose the taster sits at a revolving table with several samples of infused tea before him; he takes a sip of the tea to determine its aroma, and after rinsing his mouth with it, expectorates it. In spite of the latter precaution, possibly because of the numerous tests in any one day, more or less of the beverage is swallowed or absorbed through the mouth and may produce chronic "thein intoxication." The general symptoms of this condition vary with the dosage and susceptibility of the individual and are characterized by loss of appetite, gastric catarrh, flatulent dyspepsia, constipation, insomnia, irritability, restlessness, muscular tremblings, and gradual emaciation and in some cases also discoloration of the finger nails. Similar symptoms are observed in so-called tea and coffee topers, but fortunately the effects, like those of alkaloidal beverages in general, appear to be functional in character and pass off by removal of the cause.

Hotel, Restaurant, Saloon-keepers, Etc.—These occupations include a large number of employees, both male and female, from bell boys of the age of 14, to veterans in the army of waiters, cooks, porters, housemaids, etc.

While we have no statistical data in this country which would serve to emphasize the injurious effects on the employees as a whole, the statistics of Great Britain and of Berlin clearly indicate that this class of workers has an unusually high morbidity rate.

Dr. Gast[22] informs us that of 18,133 members of the Berlin Union, including the various classes of employees, there were 19,856 admissions a year on the sick report, with an average duration of 28.9 days, which is considerably higher than among other occupations.

Of these there were 2308 cases of diseases of the digestive organs 2087 cases of diseases of the respiratory organs, 204 of tuberculosis, 2074 cases suffered from diseases of the motor system inclusive of 1031 cases of muscular rheumatism, and 318 cases of "flat-foot" and deformities of the knee; 1585 suffered from accidents; 1417 from sexual diseases; 1016 from mental and nervous diseases; 1011 from chlorosis and anæmia; 660 from blood poisoning and cellulitis; 564 from diseases of the heart and blood-vessels, inclusive of 228 cases of varicose veins; 314 contracted occupational eczema; 212 had ulcers of the legs; and 45 suffered from chronic alcoholism and delirium tremens.

The general mortality rate was 7.16 per 1000; 45.5 per cent. of all the deaths were caused by diseases of the lungs, including consumption. The deaths from diseases of the heart and kidneys were in excess of those in other occupations. The three principal causes of death and also the undue prevalence of these diseases are doubtless influenced by the alcohol habit.

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The occupation, in general, involves long and irregular hours of work in a vitiated atmosphere, often contaminated by tobacco fumes. Many of the employees are weaklings and have inferior sleeping quarters. Prolonged standing and walking naturally favor the development of "flat-foot" and "knock- and in-knee" in the young, and of varicose veins and leg ulcers in the older class of employees. Schultes[23] has shown that of 14,000 recruits examined 12.7 per cent. suffered from flat-foot and varicose veins, mostly waiters or salesmen, while occupations involving sedentary habits furnished practically no cases. Cooks in general are exposed to excessive temperatures and this is especially true of men connected with the dining-car service, where cases of heat exhaustion are not uncommon.

Preventive Measures.—It would indeed be interesting to present American statistics for comparison. It is evident, however, from general observations that the lot of this class of employees is far from satisfactory and shorter working hours, not exceeding 8 out of 24 hours, better working conditions and pay, so as to enable them to enjoy their much needed rest, is certainly indicated.

Copious ventilation in cafes and restaurants, which permit smoking, and general sanitation of the kitchens and serving departments, will benefit the patrons and employees alike. Persons under the age of 18 should not be employed for night duty.

Domestic and Personal Service.—The United States mortality statistics of this class of wage earners including barbers, bartenders, janitors and sextons, servants and waiters and laborers not specified, show high rates from tuberculosis and pneumonia.

The rates are especially excessive in janitors and sextons, barbers, servants and waiters. The general servant, as a result of long hours and indoor life, becomes anæmic, and is liable to suffer from headache, constipation and digestive derangements, including gastric ulcer. Varicose veins and ulcers of the leg, and "flat-foot" from prolonged standing are not infrequent.

Barbers and Hair Dressers.—In view of the fact that many weaklings engage in this employment, it is not surprising that the tuberculosis death rate of barbers is rather high. Catarrhal affections of the upper air passages, digestive disorders, "flat-foot" and eczema, the latter as a result of contact with hot water and toilet preparations, are not uncommon. Razor cuts of the fingers occur occasionally, but on the whole it cannot be said that the occupation is inimical to health. The Leipsic Insurance statistics show a morbidity of 395 days per 100 members per annum and a mortality hazard of only 0.32 per cent.

Robinson and Wilson[24] inspected two bakeries in Cincinnati and found only one case of tuberculosis among 134 examined, and this man a porter had been working in the bakery only 3 months.

The conditions in the bakeries were excellent, although many of the bakers were pale, due to excessive perspiration and heat. The kneading is all done by machinery and the air is washed and humidified, before it enters the mixing rooms. The workmen are required

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to bathe and change from their street clothing into white uniforms. Promiscuous spitting is prohibited. Among 352 workers in the *confectionery and cake industries*, but two cases of tuberculosis were found. The sanitary and hygienic conditions in the four establishments visited were good in all, except that the temperature of the rooms in which the candies are packed is artificially cooled, which in summer may prove injurious on account of the sudden change from the high temperature of the street.

Out of 204 *brewers* examined four or 1.9 per cent. were tuberculous. In none of the four cases was alcoholism or occupation hazard most in evidence as a predisposing cause. The high wages, reasonable working hours, good home conditions and the high standard of sanitary conditions in the breweries, are factors tending to lessen the occupational hazards.

Of 295 males and 25 females employed in *slaughtering and meat packing*, only two cases, or 0.62 per cent. of those examined presented evidence of tuberculosis. This low percentage in the face of inimical working conditions, such as a steamy atmosphere, wet floors, extremes of temperature, is attributed to the employment of robust men, and the law prohibiting tuberculous persons from working in abattoirs.

Among 94 *restaurant employees* examined three were found to be tuberculous, although none was an open case. Six others appeared strongly predisposed to the development of the disease. Taken as a whole the group of workers presented an appearance of poorer physical condition than any other class examined. As there is nothing in the work itself that should cause tuberculosis, the investigators concluded that those who through failing health or because of drink and dissipation had failed in other work, naturally seek employment in cafés or restaurants.

Of 254 male and 377 female operatives in the *cigar industry*, only three were found to be tuberculous, although the industry has been considered to be dangerous through the inhalation of tobacco dust. However, many of the workers appeared anæmic.

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CHAPTER XVI

LIBERAL PROFESSIONS, PUBLIC SERVICE EMPLOYEES, ETC.

BY GEORGE M. KOBER, M. D., Washington, D. C.

Clergymen. Physicians. Veterinarians. Trained Nurses. Sisters of Charity. School Teachers. Lawyers. Artists. Public Speakers, Singers, etc. Musicians. Soldiers, Sailors, Pilots, etc. Fire and Police Department Employees. Stationary Firemen and Engineers. Commercial and Mercantile Pursuits. Telephone and Telegraph Service. Elevator Employees. Railway Service and Accidents. Street Railway Employees. Subway Employees. Automobilists. Draymen. Hackmen and Teamsters. Street Cleaners, Ice, Coal and Ash men and General Day Laborers. Preventive Measures.

Liberal Professions.—These include clergymen, lawyers, physicians and surgeons, teachers, nurses, artists and musicians. The morbidity and mortality statistics in all these occupations, considering the indoor and sedentary employment, are quite normal. Mortality statistics very generally indicate that clergymen have the greatest expectation of life. According to the statistics cited by Koelsch, *protestant ministers* enjoy a higher longevity than *priests*, who, however, have about the same morbidity and mortality rates as bachelors of the same age periods. Among the principal causes of death among clergymen are diseases of the heart, kidneys and apoplexy. The tuberculosis death rate in 1908 in Bavaria among this class was 1.84 against 3.07 per 1000 in the occupied male population.

The mortality of *physicians* and *veterinarians* is generally higher than in the other liberal professions, but not in excess of the occupied male population of the same ages, except in diseases of the circulatory and nervous system, and the infectious diseases. This is in part due to a life full of exposure and responsibility, and Koelsch suggests that in some instances the tobacco and drug habits may play a rôle. The tuberculosis death rate in Bavaria among physicians is quite low, viz., 1.92 against 3.07 per 1000 in the occupied male population. We have no mortality statistics concerning "*trained nurses*," and even the German statistics cover chiefly religious orders of those who consecrate their life to the care of the sick. According to Cornet and Koelsch from 55–66 per cent. of the *sisters of Charity* perish from tuberculosis, as compared with a rate of 39.6 per cent. in the general female population. Among the chief predisposing causes are doubtless the indoor life, insufficient exercise in the open air, deficient air-space of sleeping quarters, contact infections, night duty and frequent fasting.

The condition of the sisters who devote their lives to teaching is almost as unsatisfactory as regards the prevalence of tuberculosis.

The mortality rates among *school teachers*, the majority of whom are females in this country, are quite favorable, and among the German male

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teachers approximate very closely to those of the protestant clergy. The tuberculosis death rate in Bavaria was 1.3 for males and 1.55 per 1000 for female teachers, which is far below the average rate. Higher rates have been reported by Schmidt of Düsseldorf, and our American statistics also indicate that school teachers are slightly more liable to tuberculosis than members of the other learned professions.

This is doubtless due to indoor life, confinement in badly ventilated school rooms and the presence of dust. Diseases of the nervous system and uterine organs are also quite common, and teachers will do well to insist upon proper seats, absolute cleanliness of the schoolroom, fresh-air schools, or at least copious ventilation, and substitution of wet for dry methods of removing chalk markings.

The mortality rates of *lawyers* are quite favorable, although not quite as good as those of ministers, teachers and artists; their consumption rate in the British statistics is 11.6 as compared with 27.2 among their office employees. The latter, in addition to indoor work, are doubtless exposed like so many other bureau and library employees to the inhalation of very fine dust, and as a protection against this the general introduction of dust-proof file cases and cleaning by the vacuum system should be invoked.

The mortality rates among *artists* are quite satisfactory, but those of *musicians* are far below the average, due as suggested by Koelsch to a precarious existence and "a life full of temperamental conditions."

Public speakers, clergymen, singers, etc., are liable to suffer from chronic affections of the throat and paralysis of the vocal chords.

Musicians.—Pianists and violinists not infrequently suffer from spasm and fatigue neurosis of the fingers and forearms; flutists have been known to suffer from laryngeal spasm, and performers on wind instruments from spasm of the tongue and laryngeal muscles. Formerly it was held that emphysema was quite common in military musicians, but Fischer did not find a single case among the 500 brass instrument performers examined by him, and Professor Gairdner never saw a case among the Scotch bagpipe players.

Aldrich has described a peculiar neurosis, characterized by a cramp-like and burning pain, fatigue and a sense of constriction in the muscles of the right leg below the knee, caused by operating a "trap drum" by means of a pedal, which gave him free use of both hands to play other drums, triangle and "traps." The pedal mechanism required a pressure of from 5 to 25 lb. for each stroke; to play the drum required at times 180 strokes per minute, and it is perfectly conceivable how the excessive use of certain muscular groups of the right leg should be responsible for this type of fatigue neurosis. Von Wurthenau, cited by Dr. J. R. Hunt,[1] has collected 62 cases of "drummers' paralysis," which is an extensor paralysis of the distal phalanx of the thumb, occurring usually in beginners and exclusively on the left side. The condition is the result of a chronic tenosynovitis induced by a peculiar method of holding and using the left drumstick, causing a mechanical irrita-

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tion of the long extensor tendon of the thumb, and subsequent pathologic alterations in the tendon and its sheath and eventually a rupture during a paroxysm of drumming or more rarely quite spontaneously.

Dr. Hunt[1] has recorded a case which occurred in a tailor in whom the daily use of a coarse heavy needle and pressing irons evidently induced the pathological change.

Preventive Measures.—The learned professions, whose work is to a great extent mental, need physical exercise, such as golf, lawn tennis, etc., which are calculated to counteract the evil effects of a sedentary and indoor life. The benefits derived from office and house sanitation, general cleanliness, and ventilation, personal health rules as regards food and drink, and temperate habits in all things should not be underrated.

Soldiers.—The hygiene of soldiers, sailors, and members of the Police and Fire Departments is not without interest as they are usually able-bodied men and have to undergo a physical examination prior to enlistment. The health of the United States Army has steadily improved during the past three decades, as shown by a reduction in the mortality rate from 7.54 per 1000 in 1885 to 4.79 per 1000 in 1912. The majority of the men are recruited from the rural districts and in point of physique and morale compare most favorably with any army in the world. The percentage of rejection for physical defects is only 12, which is far below the average in other countries. The food, clothing, quarters and general sanitation in the United States public services are very satisfactory. The medical officers have realized long since that their chief function lies in preventive medicine, and profiting by the experience that many soldiers are lost in the making, there is no disposition to overdo the training of the recruit. Surgeon General Stokes, a few years ago, sounded a note of warning against overtaxing the officers and men in the Navy. The tendency is in the direction of steady physical gains and the percentage of recruits who breakdown during the first year of enlistment is very small. The prevention and correction of flat feet has received attention not only by proper shoes but also by proper posture and proper gait (see flat-feet).

The mortality of the enlisted men, American troops in the United States Army according to location between 1906–1913, is given by the office of the Surgeon General U. S. Army as follows:

Locality	Mean strength, exposed 1 year	Deaths	Rate per 1000
Philippine Islands	98,211	644	6.56
United States	414,783	2,038	4.91
Panama Canal Zone*	1,774	13	7.33
Alaska	8,635	43	4.98
Hawaii	14,666	51	3.48

* First Army Hospital opened in October, 1911.

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The morbidity and mortality rates are very satisfactory, except that the admission rates for venereal diseases and alcoholism are still excessive. The primary cause may be looked for in idleness, and while much has been done by athletic sports, educational methods and genteel amusements, calculated to counteract the evil effects of saloons and prostitution, more may be done by the employment of trained soldiers on extra duty for mechanical work. The admission rate for alcoholism has been reduced from 35 per 1000 in 1907 to 15 per 1000 in 1913, and for venereal diseases from 168 per 1000 in 1912 to 97 in 1913. The typhoid fever rate, the scourge of all armies has been reduced from 5.46 in 1903 to 0.05 per 1000 in 1913. This is the result of improved sanitation, but chiefly a direct effect of anti-typhoid vaccination, introduced in the United States Army in 1909.

The tuberculosis and pneumonia death rates in the United States Army have been reduced respectively from 0.95 and 0.48 in 1901 to 0.49 and 0.28 in 1913. This is largely due to the increased cubic air space in military barracks. Sir Thomas Oliver states that the phthisis death rate in the British Army was reduced from 7.82 to 2.5 per 1000 in the course of 5 or 6 decades.

Col. H. M. Dean of the Britsh Army Medical Corps, cited by Oliver, believes that the soldier's irritable heart, first described by Da Costa of this country, is largely the result of standing "at attention," with his chest advanced, walls fixed and the abdomen retracted. Such a position naturally impedes inspiration and expiration, and the blood tends to accumulate in the right side of the heart and venous system. The pulse rate is often increased from 80 to 100 and even 132 per minute, and he believes that "the mischief done to the recruit on the parade ground can be stopped by abolishing the present position of 'attention' and giving short plain instructions as to how to stand erect without constraint."

Navy and Marine Corps.—The hygiene of the public services is quite satisfactory, as shown by the following comparative mortality statistics of the United States Army and Navy:

Principal causes of death	Army rate per 10,000		Navy rate per 10,000	
	1906	1913	1906	1913
Accidents	15.7	15.3	22.1	14.2
Tuberculosis	7.7	4.9	4.4	4.5
Respiratory diseases	3.1	3.7	1.8	3.6
Digestive diseases	5.0	3.7	3.2	2.5
Circulatory diseases	2.7	2.9	3.5	2.2
Suicides	6.7	5.1	....	2.1
Genito-urinary diseases	1.4	2.1	2.1	1.9

The sailors' greatest enemies are darkness, dampness, insufficient air-space in the sleeping quarters, exposure to extremes of heat and cold, irregular

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sleeping hours, hard work and accidents, especially during storms. As a result we see an undue prevalence of injuries, diseases of the respiratory organs, rheumatic and neuralgic affections. Cases of heat exhaustion are not uncommon in stokers and engineers, especially in the tropics. Venereal diseases and alcoholism are still quite frequent, although the admission rate from alcoholism has been reduced from 5.28 per 1000 in 1907 to 3.45 in 1913. Tropical diseases naturally help to swell the general mortality rate, which, however, has been reduced from 11.85 per 1000 in 1885 to 6.22 in 1910. For further details see page 710.

Among the occupational risks in the United States Navy, Surgeon General Stokes refers to the injurious effects of carbon monoxide, which develops in gun firing in the turrets of battleships, and believes that this gas in the furnace rooms gives rise to acute poisoning, often mistaken for heat prostration. He has also called attention to the serious injury inflicted upon the retinas and optic nerves of men who have to operate strong search-lights, and has further pointed out that gunners, who use telescopes, to point the big guns in the turrets, soon fall 8 or 10 below normal vision.

Merchant Marine Service, Etc.—We have no separate statistics for sailors in this country. The United States census gives the death rate for "sailors, pilots, fishermen and oystermen" at 22 per 1000 in 1890, and 27.7 in 1900; for "boatmen and canalmen" the rates were 20 in 1890, 18.8 in 1900 and 22.9 per 1000 in 1909. Of 14,469 cases treated in the Marine Hospitals during the fiscal year ended June 30, 1914, there were 2579 general and local injuries with 25 deaths, and 11,890 cases of sickness with 431 deaths. In addition 38,757 cases were treated in the dispensaries, of which 6816 were for injuries and 31,941 for diseases. Of the total number there were 8601 cases of venereal diseases, 7219 cases of diseases of the digestive system, 3104 cases of diseases of the respiratory system, 2731 cases of rheumatism, 1361 cases of nervous diseases, and 870 cases of diseases of the circulatory system.

RATIO OF DEATHS FROM SPECIFIC CAUSES IN THE U. S. MERCHANT MARINE SERVICE

	1906	1914
General diseases	51.52	48.23
Diseases of the nervous system	4.87	6.39
Diseases of the circulatory system	11.16	14.53
Diseases of the respiratory system	9.13	9.25
Diseases of the digestive system	5.88	5.28
Genito-urinary system	6.09	7.93
Injuries	9.13	5.52
From all other causes	2.22	2.86

The mortality rate of the British Merchant Service given by Oliver[2] for 1898–99 was 9.6 per 1000, of which 7.4 were caused by injury and 2.2 from disease. The corresponding rate in the Royal Navy for 1899 was 4.9 per 1000 made up of 1.35 from injury and 3.56 from disease, showing what can

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be done by rigid discipline, and prompt medical attention in the prevention of fatal accidents. The same author states that cases of thoracic aneurism, as a result of hard work, sudden strain and syphilis, also of violent knocks and bruises of the chest received during gales, are quite common.

The occupational hazards of *pilots*, *fishermen* and *oystermen* do not differ essentially from those of sailors. Exposure to all kinds of weather, long hours, irregular meals and accidents from sudden squalls naturally render an otherwise healthful out-door occupation dangerous.

The members of the *Life Saving Service*[3] encounter even greater occupational risk than pilots and fishermen. The average age of *station keepers* in 1910 was 51 years and of *surfmen* 38 years. The fatal accident mortality in 1880, in a force of 1167, was 5.14 per 1000 and in 1910 with a mean strength of 2204 it was only 0.45 per 1000.

Fire and Police Department Employees.—The men employed in the fire department, like all other workers, exposed to excessive heat, sudden changes in temperature, wet and cold are predisposed to rheumatic, neuralgic and catarrhal affections, muscular cramps and diseases of the respiratory organs.

In addition to the injurious factors common to all firemen they are not infrequently exposed to the inhalation of hot smoke, carbon monoxide and illuminating gas, nitrous fumes and other toxic vapors from acids, chemicals, celluloid, benzine, turpentine, etc. Cases of asphyxia, pulmonary œdema and pneumonia are not uncommon. Accidents and electric shocks, are also quite frequent. The fatal accident rates of the chiefs of batallion in the New York City fire department between 1902–1911 according to Hoffman,[4] was 8.4 per 1000 and that of the firemen 2.4 per 1000. The men employed on the police force are engaged in out-door work and the duties are not unlike those of soldiers in time of peace. The chief occupational risks are exposure to inclement weather, long hours, irregular sleep and meals.

The following comparative mortality statistics are compiled from data furnished by Hoffman:

Principal causes of deaths	New York Fire Dept. 1902–1911, force exposed per annum 37,526, rate per 10,000	Chicago Police Force 1901–1910, force exposed per annum 34,087, rate per 10,000
Accidents	23.2	17.6
Pneumonia	9.3	15.5
Tuberculosis	6.9	12.9
Urinary diseases	5.3	10.0
Heart diseases	4.8	14.7
Digestive diseases	3.7	6.5

The mortality rates from pneumonia and tuberculosis in the Chicago police force are especially excessive, which is doubtless due to climatic and racial conditions in that city.

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Stationary Firemen and Engineers.—The men employed in power plants notably the firemen, coal heavers and laborers, are exposed to excessive heat and sudden changes in temperature, especially when the workmen step out-doors to cool off. There is also more or less exposure to dust from ashes and coal, and escaping gases and steam. The work, unless automatic stokers are employed, is fatiguing and is often carried on in dark, damp and dirty quarters. The mortality from tuberculosis is about 11 per cent. and the accident rate about 12 per cent.

The men engaged in attending furnaces in connection with foundry work, kilns and drying rooms, enamelling, japanning, baking ovens, etc. are exposed to similar occupational risks. In some industries there is an added danger from poisonous dust and fumes, and exposure to intense heat and light.

Working conditions should be improved by adequate exhaust fans movable screens, mechanical contrivances, eye protection against intense heat, light and foreign bodies. Shower baths and facilities for personal cleanliness are essential.

Commercial and Mercantile Pursuits.—There are two groups of employees in commercial pursuits which deserve consideration. The first group, according to Chajes,[5] includes the office employees, bookkeepers, cashiers, stenographers, typists, travelling salesmen, clerks, stockclerks, floor walkers, etc.; the second group includes porters, shipping clerks, delivery wagon drivers, messengers, laborers, etc.

The German Insurance statistics from 1903–1905 show that of 215,981 male employees there were 76,334 cases of illness or 35.38 per cent. per annum. Of 114,824 female employees there were 48,733 cases of illness, *i.e.*, or 42.44 per cent. The average for both sexes was 38.8 per cent. which is about 3 per cent. higher than the general average in other occupations. The prevalent diseases among the male employees were diseases of the respiratory and digestive organs and diseases of the nervous system. Among the female employees, anæmia and chlorosis came first with a morbidity rate of 30 per cent.; next diseases of the respiratory and of digestive organs; followed by diseases of the nervous system. About 29.5 per cent. of those reported sick had sustained accidents, which are especially common among the porters and packers and in the stock and shipping departments. Near-sight, caused by defective lighting, and deformities of the knee, "flat-foot," and varicose veins are not uncommon.

About 19 per cent. of the morbidity among the office and store force was due to diseases of the lungs; the percentage among the porters, packers, etc., was betwen 21 and 29, probably on account of additional exposure to dust; 41 per cent. of all the deaths were caused by tuberculosis. Among the diseases of the digestive organs with a morbidity rate of 7–11 per cent., gastric and intestinal catarrh, chronic constipation, gastric ulcer, and hemorrhoids were the most frequent. Rheumatism, anæmia, neurasthenia

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and other nervous affections are not uncommon. Occupation neuroses are occasionally observed in stenographers and typists.

Travelling salesmen often contract the alcohol habit. Peddlers and small shopkeepers have a very high tuberculosis death rate in Bavaria, 8 per 1000 against 3.07 in the occupied male population (Koelsch).[6]

The long hours, indoor life, exposure to vitiated air, uneven temperature, drafts and sedentary habits in the office force naturally favor the development of pulmonary disorders. These same factors, together with the mental strain in accountants, typists and stenographers, also predispose to anæmia, chlorosis, malnutrition and diseases of the nervous system. Cold lunches, improper food, hasty eating, sedentary or standing habits and many of the conditions incident to indoor occupations, are doubtless responsible for the undue prevalence of diseases of the digestive organs.

Preventive Measures.—Commendable progress has been made in the sanitation of mercantile establishments, but much more needs to be done in the way of ventilation, heating, lighting and general cleanliness. All sweeping and dusting should be done by the vacuum system. Vestibule doors should be provided in winter to avoid cold drafts. The law makes provisions for seats for female clerks and they should be encouraged to use them.

Stores should not be open for over 9 hours a day with 1 hour for lunch and rest. Large establishments should provide rest rooms and hot lunches at reasonable prices. Female employees deserve special consideration; they are usually underpaid, and as a result the home environments are not conducive to health.

Telephone and Telegraph Service.—The telephone service is rapidly growing and is of special interest because it affords employment to a large number of females. The occupation involves indoor life, concentration and alertness of mind, and many vexations which cannot fail to exert an injurious effect upon the operators.

Statistical data is still lacking, but general observation indicates that the switchboard service predisposes to the development of neurasthenia and hysteria. Opinions as to the effect of the occupation upon hearing differ very widely; some authors hold that it leads to impairment, while others contend that the sense of hearing is rendered more acute.

A few cases of paralysis of the vocal chords have been reported and attributed to the excessive use of the voice and constrained position in telephoning. A more important group of injuries is caused by sudden and violent acoustic effects upon the auditory nerve of the person using the receiver. This phenomena may be caused by entrance of a high-tension current into the telephone circuit, by breakage of a power or light cable, or during an electric storm. According to Bernhardt[7] in some instances these violent acoustic bangs have produced rupture of the ear-drum. This accident must be very rare, however, as I have been unable to find similar cases in the literature.

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In the majority of cases, a slight shock from low voltage simply produces a psychic shock and the frightened operators sometimes become quite hysterical.

M. Capart's[8] extended experience leads him to conclude that the normal operations of the telephone produce no injurious effects. But abnormal acoustic effects are very liable to develop profound symptoms of hysteria and aggravate chronic affections of the ear. The presence of impacted ear wax was found by Castaneda[9] to be the cause of frequent attacks of nausea and dizziness in a telephone operator. Koelsch[10] informs us that in Germany during the year 1907, eight telephone operators were injured by the transmission of high-tension currents, seven by the induced current, and two others by the effects of lightening.

In the Chicago Telephone Co., according to Dr. J. D. McGowan,[11] there were in 1 year 473 cases of shock reported, two of which resulted in death. Most of the serious cases occurred in linemen.

Dr. McGowan also refers to the accidents liable to befall linemen, such as fractures, dislocations, sprains, wounds, contusions, and nail punctures, and the frequency of septic infections of wounds, caused by copper wire. It is difficult to conceive why copper or verdigris, which is so much feared by the workmen both here and abroad, should be held responsible for septic conditions of wounds which are of bacterial origin. This class of employees is especially liable to electric shock, burns and other injuries from crossed wires, etc., which have been considered in another chapter, see page 402. Hoffman's statistics based upon 205 deaths among electric linemen show a mortality of 48.3 per cent. from accidents 18.5 per cent. from tuberculosis, 6.3 per cent. from other lung diseases, 6.3 per cent. from heart disease, 3.9 per cent. from digestive diseases, 3.4 per cent. from urinary diseases. Workers in cable vaults are also often exposed to gas poisoning. (See page 416.)

The occupation of telegraphers involves many of the risks already referred to, except that the telegraph operators, as a result of over-exercise of certain groups of muscles, are liable to develop a fatigue neurosis of the muscles of the hand and forearm, which is analogous to "writers' cramp."

Preventive Measures.—There is no doubt that the occupation of both telephone and telegraph operators involves a severe strain upon the nervous system, which can be greatly mitigated by favorable working conditions, shorter working hours, and by kind and considerate treatment on the part of the general public. The female operators, because of the strenuous character of the work, are entitled to special protection and should not be obliged to work over 8 hours out of the 24, with reasonable intervals for rest, which should be taken in cheerful, airy and comfortable rest rooms.

In the matter of accident prevention, Dr. McGowan states that, while shocks are common, severe shocks causing deaths are now rare in comparison to what they were 10 years ago, owing to the various safety devices, the greater care in supervision and greater caution on the part of the employee.

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The large number of accidents from electric shock in Chicago, as compared with the German statistics, shows that more remains to be done to prevent the transmission of high-tension currents by the abolition of overhead wires, and the general introduction of safety devices for the prevention of the entrance of strong currents of atmospheric electricity.

Operators and all others using the long distance wires during electric storms should handle the receiver with a silk glove or handkerchief. Excellent safety devices have been introduced for the prevention of slight shocks from a low voltage, caused by the old-time method of violent ringing.

The majority of telephone operators in the central station in the City of Washington are females; they are on duty for 9 out of 24 hours, with 1 hour for complete rest, and intermissions of 10 minutes after 2 hours of work.

The company has provided rest rooms, lunch rooms and an emergency room. The working conditions are favorable, and all vexatious patrons are promptly switched over to a calm and tactful operator, who adjusts the difficulties and thus spares both parties from a nervous strain.

Elevator Employees.—A large number of persons are employed as elevator conductors. The accident liability in this occupation is by no means insignificant and numerous fatal injuries, the result of carelessness or direct violation of rules, such as open doors or sudden starting, are constantly recorded.

Among the non-fatal injuries may be mentioned fractures of the lower extremities, pelvic bones and spine, the result of falls through an open door down the elevator shaft, while crushing injuries of the foot are not infrequently caused by being caught in starting between the door and shaft. Fatal injuries and contusions of the chest often result from carelessness during the oiling or inspection of the running gear, and the head injuries are usually caused by falling objects from above. Minor injuries, such as contusions and fractures of fingers or bones of the hand, may result from being caught between the doors in the attempt to close them. Lacerations of the hand may occur as a result of a slipping wire rope, which is also the cause of painful callosities and contracted tendons in the palm of the hand.

"Flat-foot" and varicose veins, the result of prolonged standing, and catarrhal, rheumatic and neuralgic affections, caused by drafts and uneven temperature, are frequently observed.

Preventive Measures.—Reasonable working hours and strict enforcement of the rules governing the elevator service.

Railway Service.—The employees in the railway service, apart from liability to accidents, lead a life full of responsibility and hardships. This, together with irregular habits as regards work, sleep and meals, and exposure to dust, drafts, etc., increases their liability to sickness in spite of the fact that generally only able-bodied men are employed.

According to Rüdlin[12] of 280,098 employees in the Prussian-Hessian system in 1905, the morbidity rate was 41.97 per cent. with an average dura-

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tion of 26.69 days. The German statistics prior to 1890 show a morbidity rate of 48 per cent. The train hands, dispatchers, switchmen, track walkers and gatemen show the greatest amount of sickness and the station and office force the least. Diseases of the digestive organs, diseases of the respiratory system, rheumatism and diseases of the nervous system appear to predominate.

Schwechten[13] reports a notable decrease in the tuberculosis death rate, which has declined from 34.8 per 10,000 of employees in 1878–82 to 16.3 per 10,000, a reduction of 53 per cent. While organic diseases of the nervous system, railway spine, insanity and apoplexy have decreased, an increase in functional disorders, such as neurasthenia, hysteria, etc., is noted.

The undue prevalence of rheumatic, catarrhal and neuralgic affections is probably accounted for by sudden changes in temperature to which train hands are especially exposed during the cold months.

Dr. Edsall[14] upon the authority of Dr. Latta, Railway Surgeon of the Penna. R. R. Co., calls attention to the frequency of right-sided cases of sciatica in locomotive engineers. This was traced to the fact that some of the men are in the habit of sitting sidewise on the bench of the right-hand side of the engine cab and, in the constant jolting, the weight of the body is continually thrown violently on the right thigh and buttock. The number of cases was reduced and the special tendency to right-sided cases eliminated by simply cutting off the forward part of the bench, so that the engineer had to sit squarely facing forward.

Disorders of the digestive system are doubtless influenced by irregular meal hours, and the character of food and drinks. Iced or very cold drinks are especially harmful, and since the months of July and August furnish the greatest number of cases of gastric catarrh, this causative factor may at least be assumed.

Hedinger[16] called attention to the fact that only 8 per cent. of the German locomotive engineers have normal hearing, while 67 per cent. of the engineers and 30 per cent. of the firemen, and 14.5 per cent. of the track walkers had defective hearing. The percentage in all increased with the length of service. The most common affection was catarrh of the Eustachian tube, and internal ear, probably caused by frequent exposure to abrupt changes in temperature.

Hoffman's[16] statistics based upon 537 deaths among brakemen show a mortality from accidents of 63.3 per cent. from tuberculosis 11.9 per cent., pneumonia 3.2 per cent., digestive diseases 3 per cent., heart disease 2.6 per cent., nervous diseases 2 per cent., apoplexy and paralysis 1.5 per cent.

Railway Accidents.—The fatal accidents to railway employees have been gradually reduced from 9.52 per 1000 in 1890 to 4.78 per 1000 in 1909. The following table compiled by the Prudential Insurance Company of America is of special interest.

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CAUSES OF FATAL ACCIDENTS TO TRAINMEN, 1901–1909
(Employees exposed 1 year 2,363,077)

Cause	Total accidents	Rate per 10,000
Falls from trains	3710	15.7
Collisions	3202	13.6
Struck by trains	3104	13.1
Derailments	1974	8.4
Coupling or uncoupling	1874	7.9
Other movements of trains	1624	6.9
Jumping on or off	943	4.0
Overhead obstructions	634	2.7
Breaking down of equipment	199	0.8
Parting of trains	154	0.7

SUMMARY OF ACCIDENTS RESULTING FROM COLLISIONS AND DERAILMENTS FOR THE 10 YEARS ENDED JUNE 30, 1913*

Year	Number	Persons		Damage to road and equipment, and cost of clearing wrecks
		Killed	Injured	
1904	11,291	1,018	10,244	$9,383,077
1905	11,595	1,064	11,949	9,711,656
1906	13,455	977	12,686	10,659,189
1907	15,458	1,291	16,236	12,865,702
1908	13,034	728	12,834	10,183,660
1909	9,670	606	9,560	7,480,203
1910	11,779	773	12,579	9,823,958
1911	11,865	785	11,793	9,851,780
1912	13,698	772	15,096	11,527,458
1913	15,526	791	14,565	13,049,214

Preventive Measures.—Every effort which will promote the health and comfort of passengers in the way of temperature, ventilation, light and mitigation of the dust evil, cannot fail to prove beneficial to a large number of employees. The sanitation of passenger coaches is still defective, in the matter of cleaning, which should be done more frequently and thoroughly. The vacuum system is especially indicated for upholstery. Leather or some impermeable material should replace plush covers. The temperature in cars during the cold months should be maintained between 70° and 75°.

It is generally conceded that safety in railroad operation is the result of: (1) Properly constructed and maintained track signals and rolling stock. (2) Careful rules and regulations. (3) Carefully selected and trained em-

* Figures for number of persons killed and injured for years prior to 1911 are restricted to passengers and employees on duty. (27th Annual Report of the Interstate Commerce Commission, Washington, D. C. 1913.)

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ployees, of sober habits, who faithfully discharge their duties. (4) Intelligent and comprehensive supervision.

These cardinal principles are carried out by the management of the Pennsylvania Railroad Lines, with the result of having the best record in accident prevention in 1912. The Company maintains its rolling stock with a large percentage of steel cars in a high state of efficiency and has introduced safety devices in the shops and roundhouses where over 41,000 men are employed. In 1911 the Company appointed 35 safety committees who made 17,333 formal recommendations, of which 13,861 were acted upon, one of the results being that by the expenditure of $99,753 for safety guards, mainly in shops, the serious accidents to shop employees have been reduced from 5.4 per 1000 to 3.2 in 1912. The Company maintains 105 hospitals, and employs an efficient medical corps not only for emergency and hospital work, but also for the instruction of all employees in first aid. During the year ended August 31, 1913, 195 lectures and demonstrations were given, particularly at the larger shops. All stations, shops, engine cabs, cabin cars, etc., are equipped with "first-aid" kits.

Street Railway Employees.—The hygiene of this occupation does not materially differ from that of the general railway service. The working hours of the traffic force are unusually long, the shifts are irregular and involve both night and day work.

Professor Weyl,[17] with his characteristic thoroughness, presents statistical data bearing upon the accident and morbidity liability of this class of workers in Germany.

In 1905 there were reported among 60,940 employees 4253 accidents or a rate of 69.73 per 1000 full-time workers. Of this number 31 proved fatal, 35 resulted in permanent total disability, 293 in partial permanent disability, and 104 in temporary disability. A majority of the serious injuries were caused by falls and being run over.

The inspectors and overseers of the traffic lines furnished the largest percentage; next came the employees in the mechanical department, followed by the motor men and conductors. The same relative frequency was noted in the morbidity rates, except that the "extra force," which is made up of inexperienced men, furnish the highest rates.

The morbidity statistics, which are based on a membership of 8523, show that there were 6471 cases of sickness with 88,149 days' loss of work. Colds, influenza, tonsillitis and rheumatism were the cause of illness in 2411 cases; digestive disorders furnished 1248 cases, diseases of the respiratory system, inclusive of consumption, 611. The mortality rate from consumption, which was especially high in the office and temporary force, was 22.6 per cent. There were also 265 cases of mental and nervous diseases; 376 cases of skin diseases; 222 cases of ear, nose and throat affections; 161 cases of diseases of the eye; 89 cases of heart disease; and 54 cases of varicose veins; cases of "flat-foot," hernia and frost-bite are not uncommon.

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In view of the fact that the employees are primarily picked men, the morbidity rates appear to indicate that the same injurious factors pointed out in connection with the general railway service also operate in this branch.

Dr. Schwarze[18] emphasizes the strain upon the eyes and ears, the development of "flat-foot" and varicose veins from prolonged standing and the undue prevalence of chronic conjunctivitis, from exposure to dust and drafts as incident to the occupation. While the occupation predisposes to nervous affections, the real cause of neurasthenia is often not at all connected with the service. Motormen who almost constantly stamp on the gong pedal often develop neuroses of the right leg. Schwarze refers to occasional cases of burns from the electric current, and to the comparative rarity of injuries from electric shock, having observed but one case during 10 years. As causes of hernia he mentions the faulty position of the motormen in the use of the brake, and the habit of the conductors in leaning too far back over the rear platform, to which may be added the muscular strain involved in jumping on and off the cars.

Preventive Measures.—While it cannot be said that this occupation is intrinsically dangerous to health, much could be done to diminish exposure to the elements and dust by inclosed platforms. Adjustable seats for the motorman should be provided. The clothing should be adapted to climate and seasons. The establishment of reasonable working hours, and of lunch rooms, where hot soups, coffee, tea, etc., are served, would do much toward the prevention of the alcohol habit and of accidents.

Subway Employees.—We have as yet no statistical data to justify positive conclusions as to the injurious effects of employment in the subway service. Dr. Sutherland determined that the proportion of carbon dioxide in the air of subway stations and cars varies from 4 to 20 parts per 10,000 volumes as compared with 2.8 in the outer air.

Dr. G. A. Soper[19] of New York has shown that the subway dust contains 61.3 per cent. of iron, mostly in the form of fine platelets; 21.9 per cent. of organic matter of animal and vegetable origin; 15.6 per cent. mineral matter, mostly silica; and 1.3 per cent. of oily matter.

The average amount of dust was 61.6 mg. and the maximum weight was 204 mg. per 1000 cubic meters of air. The excess of dust in the subway air over the outer air averaged 47 per cent. Dr. Soper found that magnets hung up in the subway, collected more particles of iron than magnets of the same size and strength hung up in an iron foundry or in a dry grinding and polishing establishment. From samples taken it was estimated that in every month 25 tons of iron and steel are ground off the rails, brake-shoes and wheels on the 21 miles of subway. The greatest wear and tear falls upon the cast-iron shoes of the powerful brakes, amounting to 1 ton per mile a month.

The physical examination of the men[20] revealed an excessive amount of dry pleurisy without pain or other physical discomfort, the proportion being 53 per cent. against 14.5 per cent. among men not engaged in subway work.

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Congestion and inflammation of the upper air passages were also prevalent. The drafts and changes of temperature at or near the stations and the character of dust are doubtless injurious factors.

Preventive Measures:—It is reasonable to assume that the factors mentioned are inimical to the health of employees, who are exposed for several hours daily to the inhalation of dust, which is known to produce structural changes in the lungs. No effort should be spared to reduce dust production and to mitigate its effects by ventilation and copious oiling of the road beds.

Automobilists.—The hygiene of automobilists is still in its infancy and we have no statistical data to justify positive conclusions as to the influence of exposure to fine road dust, smoke, and other products of gasoline combustion upon the habitual users of automobiles. Dr. Delavan[21] has pointed out that dust and smoke, temperature, air pressure and winds are fruitful causes of inflammatory conditions of the nasal cavities, pharynx, larynx, trachea, bronchi and lungs. It can be readily understood how these causative factors are intensified during speeding, since a speed of 30 miles will create, even in a calm atmosphere, currents equivalent to a stiff breeze. This alone is calculated, especially in cold weather, to abstract an undue amount of animal heat and prediposes to congestions of the internal organs, while dust and fumes will serve to aggravate the evil effects.

Cases of gasoline poisoning have been reported by Dr. Box,[22] in chaffeurs who were maneuvering a motor car in a closed garage. Similar cases are mentioned by Oliver[23] and also cases of carbon monoxide poisoning, from defective exhaust pipes.

Affections of the eyes and skin, caused by exposure to the sun and dust, are not infrequent. Sciatica, neuralgia, muscular rheumatism and numbness in the lower extremities are prevalent, and attacks of pseudo-angina have been observed after violent efforts in cranking. Constant and excessive pressure with the right foot has been known to produce sciatica and lumbago. It is highly probable that the nervous strain, combined with constant and rapid concussions, tends to produce similar diseases of the nervous system as are observed in locomotive engineers.

Borattaue[24] mentions that injurious effects are produced in all persons suffering from diseases of the respiratory, circulatory and female genital organs; and Paul le Gendre[25] reports cases of sudden death in chaffeurs from heart lesions. The liability to accidents from collisions and otherwise is also very considerable in this age of speed. Colles's fracture the result of back fire is notoriously common.

Preventive Measures.—Dustless streets and roads, rules limiting the speed to 12 miles or less per hour in cities, and to 20 miles on country roads, careful traffic rules in cities, proper clothing and protection of the eyes by suitable glasses, wind shields, and the habit of breathing through the nose, are also important safeguards, while automatic sparking blocks will diminish the number of Colle's fractures.

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Draymen, Hackmen, and Teamsters.—During the census year of 1900 the number of drivers and teamsters in the U. S. was 532,632; the mortality rate from consumption was 2.6 per 1000, and the death rate from other diseases of the lungs was 1.7 per 1000. (See also page 753.)

The industrial insurance statistics cited by Hoffman[26] include 3850 deaths, of which 999 or 25.9 per cent. were caused by consumption, 483 or 12.5 per cent. from pneumonia, 56 or 1.5 per cent. from bronchitis and 93 or 2.4 per cent. from other diseases of the lungs making a combined death rate of 42.4 per cent. from diseases of the respiratory organs, against an expected rate of 26.5 per cent.

The mortality rate from diseases of the heart and arteries was also excessive. While exposure to road and municipal dust, inclement weather, and hard work, especially in the draymen and teamsters, are doubtless important predisposing factors, the effects of the alcohol habit cannot be ignored.

The death rate from accidents and injuries in this class of employees in 1900 was 1.33 per 1000. Only six of the other 27 occupations tabulated furnished a higher rate.

Preventive Measures—The undue prevalence of diseases of the respiratory organs plainly indicates that dust plays an important rôle; hence no effort should be spared to reduce the amount of road and street dust, primarily by proper construction of the highways and next by frequent cleaning of streets and oiling of roads.

Street Cleaners, Ice, Coal and Ash Men and General Day Laborers.—Although these occupations are carried on largely in the open air, foreign statistics show that this group is subject to the highest morbidity and mortality rates, especially from diseases of the respiratory organs. The accident rate is also excessive, and diseases of the heart and arteries as a result of hard work are likewise quite common.

The dirty character of the work predisposes to diseases of the skin and septic infections. Rheumatism as a result of exposure to the elements is not infrequent, and the undue prevalence of diseases of the digestive system is doubtless influenced by the scale of wages and character of food. Our American statistics are limited to street cleaners and the evidence gathered by Hoffman[27] from Medical Journals indicates that about one-third of the 5000 street cleaners, in 1907, in New York, were infected with tuberculosis, in spite of the fact that these men, previous to employment, were examined by Civil Service Surgeons and pronounced free from organic disease. The men employed on the East side, where the streets are in a more filthy condition, suffered more from diseases of the nose, throat and lungs, than the sweepers of the West side.

The more accurate data furnished by Hoffman[27] are based upon the industrial insurance statistics, which deals with 180 deaths among street cleaners; of this number 33 or 18.3 per cent. died from consumption, and

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a like number perished from pneumonia and other diseases of the lungs, making a combined mortality rate of 36.7 per cent. from diseases of the respiratory organs. The Bavarian statistics cited by Koelsch[28] show that the tuberculosis death rate in this group of employees is 83.1 per 1000 living, or 27 times higher than in the average occupied male population. In England it is 5–6 times higher. On the other hand, we have the studies of Augustus Gay, cited by Edsall,[29] on nightmen, scavengers and dust men showing that they presented an unusually good average health. The observations made in the so-called dust women of London, who are engaged in picking from the city refuse such articles as rags, twine, paper, old leather and rubber, bones, coal, cork, broken glass, etc., led Oliver[30] to conclude that, in spite of the extremely dusty character of the work, the women become apparently immune to its possible evil effects—all of which indicates that more intensive studies are needed. Kryz[31] has recently called attention to the sharp, angular and irritant character of dust from ashes.

It is difficult to determine how much social economic factors have to do with the excessive rates of the German day laborers, but they are perhaps no more potent than in the same group of laborers elsewhere.

Exposure to the elements, dust, and tubercle bacilli are predisposing factors everywhere. While the danger of infection from street dust has probably been overstated, because the tubercle bacilli perish under the influence of sunlight, they have been found in street dust by Dr. Robertson in Birmingham and Martin of Leipsic. Kristen of Breslau has shown that they may survive from 3–8 days in such material.

Preventive Measures.—It is hoped that anti-spitting ordinances and the modern system of cleaning streets, by means of sprinkling and sweeping machines, will be generally adopted and thus reduce the dangers of dust inhalation to a minimum. The wet process is equally applicable to the unloading of coal, the collection of ashes, etc.

All dry refuse should be moistened and removed under cover. A higher scale of wages, better food and better housing conditions are indicated, and will do much toward the prevention of the general misery of this class of wage earners.

Fiske presents tables of occupational distribution of physical disability in the personnel of the U. S. Navy.[32] In a personnel of 62,251 officers and men for a period of six years the non-effective rates per 1000 caused through sick days, invaliding or death are as follows: Officers 31.88; midshipmen 35.01; electricians 32.61; engine room force 37.74; fire room force 45.02; all other artificers 40.23; clerical force 31.16; culinary force 40.50; hospital corps men 48.90; marines 58.17; musicians 26.15; prisoners 16.20; apprentice seamen 110.84; ordnance 26.75. All other in the seaman branch 35.55. Practically over 11 per cent. of apprentice seamen and nearly 6 per cent. of the marines were unavailable for duty, while all the other groups show a disability rate of less than 5 per cent.

The admission rate for *affections of the eye* varied from 10.97 for the whole service to 154.60 for midshipmen. The admission rate for *diseases of the ear* was especially high in apprentice seamen, marines and coal passers. The admission rate for *mental diseases* was relatively high for apprentice seamen, fire room force, hospital corpsmen and marines.

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The rates for *neurasthenia* were 8.84 for officers, 4.95 for yeomen, 3.58 for hospital corpsmen and 3.05 for musicians. The admission rate for *skin diseases* was lowest (16.56) in hospital corpsmen, next (19.16) in yeomen and highest 177.36 for midshipmen. In the latter class Surgeon Fiske believes that the swimming pool, carelessness in using others towels, soap, athletic clothes and infected gymnasium apparatus deserve attention. The admission rate for *respiratory diseases* for apprentice seamen was 103.07 per 1000 against an average of 22.80 for the service at large. The admission rate for *tuberculosis* for apprentice seamen was 7.04 per 1000 against 5.26 for the whole service. The death rate from all respiratory diseases for apprentice seamen (years 1909–1914) was 7.58 per 1000, approximately 50 per cent. higher than for any other occupational group of the Navy. In addition to unfavorable climatological naval training stations, mentioned by Fiske, it is possible that close quarters favor contact infections.

Midshipmen have a very high *injury rate*, 204.52. The fire room force shows a rate of 100.93; engine room force 83.52; electricians 62.87; Surgeon Fiske believes that these rates warrant remedial measures in athletics and safety devices in shopwork, etc. *Rheumatic and heart affections* are especially high in recruits, coal passers, marines and apprentice seamen.

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LIST OF INDUSTRIAL POISONS*

(Translated by Wm. H. Rand, M. D.)

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
ACETALDEHYDE, ETHYALDEHYDE, CH_3COH: A colorless, very volatile fluid, of pungent odor.	Manufacture of vinegar; silver mirror manufacture.	In the form of vapor, through the respiratory organs and mucous membranes.	Irritation of the mucous membranes of the nose, larynx, and bronchi; irritation of the mucous membranes of the eyes; acceleration of the heart's action; profuse night sweats.
ACRIDINE, $C_{13}H_9N$: Crystallizing in colorless needles; contained in anthracene.	Organic dyes industry.	Exerts effect in any state of aggregation on skin and mucous membranes.	Irritation and inflammation of skin and mucous membranes; severe burning and itching of the skin; violent sneezing.
ACROLEIN, C_2H_3COH: A colorless, very pungent smelling fluid, of fiery taste.	In the frying out of fat and fat-containing material, *e.g.*, in bone rendering plants; oilcloth and linoleum factories; varnish-boiling shops; tallow-rendering establishments; soap factories (sulphuric acid process), and stearic-acid factories.	In vaporous form, through the organs of respiration and the mucous membranes.	Itching in the throat; irritation of the eyes, exciting lachrymation; conjunctivitis; irritation of the air passages, bronchial catarrh.
AMMONIA, NH_3: A colorless gas of sharply pentrating odor.	Coke ovens; mirror-silvering industry; coating iron plate with tin or zinc; manufacture of solidified ammonia, sulphate and chloride of ammonium (sal ammoniac) from ammonia water; manufacture of the carbonate of soda and of orselle dye-stuffs; dyeing industry; sewer cleaning; manufacture of bone black; gas plants; varnish and lacquer manufacture; tanning; beet-sugar manufacture; manufacture of ice; refrigeration plants.	In gaseous form, through the organs of respiration. Seldom pure, mostly in combination with other gases. Immediate effect on the conjunctiva and the cornea.	A proportion of more than 0.15 per cent. of ammonia in the air immediately causes an irritable condition of the mucous membranes. Chronic bronchial catarrhs are especially liable to follow long-continued inhalation of small quantities of the gas diffused in the air. From these are to be discriminated the acute conditions of transient illness: Intense irritation of the respiratory organs; violent sneezing; lachrymation, redness of the eyes, inflammation of the cornea and of the conjunctiva increased secretion of saliva; burning in the pharynx, and a sense of constriction in the larynx; paroxysmal cough, with secretion of tenacious, viscid, even bloody, mucus; embarrassment of respiration, attacks of suffocation; vomiting of serous masses; ammoniacal odor of the perspiration; retention of urine, which may last many hours and even 2 or 3 days; acute inflammation of the respiratory organs, and scattered areas of inflammation in the lungs, in severe cases, a fatal outcome. Protracted breathing of small quantities is apt to cause chronic bronchial catarrh.

Special measures of relief: Immediate removal from the poisonous atmosphere; artificial respiration; inhalation of steam; faradic stimulation of the phrenic nerve; free bloodletting; in case of obstinate spasm of the glottis, tracheotomy.

* Prepared by Prof. Dr. Th. Sommerfeld and Dr. R. Fischer, and Edited by Permanent Advisory Council of Hygiene of the International Association for Labor Legislation. Published in Bulletin No. 100, Bureau of Labor, Washington D. C., May, 1912.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
AMYL ACETATE, $C_5H_{11}CH_3CO_2$: Zapone, a solution of celluloid in amyl acetate and acetone.	Zapone lacquer used as a lacquering agent in metallic ware and jewelry factories; manufacture of metallic wire for incandescent electric lamps; oilcloth manufacture.	In the form of vapor through the respiratory organs.	Nervous symptoms; headache; fullness of the head; giddiness; nausea; numbness; disturbances of digestion; palpitations of the heart.
AMYL ALCOHOL, $C_5H_{11}OH$: A colorless, oily fluid, of very sharp taste and penetrating, disagreeable odor.	Manufacture of fruit essences, nitrite of amyl, valeric acid, and aniline dyes; rectification of spirits.	In the form of vapor through the organs of respiration.	Congestion of the head; headache; oppression of the chest; irritation of the air passages.
ANILINE, $C_6H_5(NH_2)$: A colorless oil which acquires a tint on exposure to air and light. Like aniline, all other amide compounds of benzol and its homologues, as toluol, naphthaline, xylol, etc., are poisons. Especially should be mentioned alpha and beta naphthylamine, benzidine, tolidine, paranitraniline, the diamines (phenylene and tolylene diamine) as well as the alphyl and aryl compounds of aniline, like their homologues (dimethyl and diethyl aniline, diphenylamine, etc.).	Manufacture of aniline and its derivatives, as well as of aniline dyes; manufacture of photographic materials and the like.	Absorption through the skin, by direct contact or by saturation of the clothing; through the digestive organs; absorption through the respiratory organs as volatile particles and impalpable dust.	The toxicity of the separate products is very different in degree; the para compounds are usually more poisonous than the ortho and meta compounds. **Acute Poisoning.**—(*a*) *Mild cases:* Pallor of the skin and mucous membranes, with slight cyanosis; a feeling of weariness and weakness; head symptoms—vertigo, reeling, unsteady gait; deficient elasticiy of movement; slow, labored speech; irritability (aniline "pip"); condition of slight inebriation, with loquacity, gaiety, and defective power of orientation; loss of appetite, constipation, and tense, rapid pulse. (*b*) *Severe cases:* Dark blue to swarthy cyanosis; formation of methemoglobin; bounding pulse; "air-hunger," with great frequency of respiration; lowering of sensibility; obliteration of the reflexes; sometimes vomiting, strangury and bloody urine. (*c*) *In the most serious cases:* Sudden prostration; cold, pale skin, blue lips, nose and ears; diminution and even extinction of sensibility; moist, cold skin; small pulse; death in a comatose condition, sometimes after antecedent convulsions. **Subacute and Chronic Poisoning.**—Anæmia; slowing of the pulse; disorders of digestion, such as eructations, loathing of food, vomiting, diarrhea, and eczematous and pustular eruptions on various parts of the body, especially on the scrotum; nervous symptoms, as general debility, headache, ringing in the ears, vertigo, unrestful sleep, disturbances of sensibility, often also of motility; spasmodic muscular pain. Subacute and chronic poisonings are very rare. Anæmia and retarded pulse are early symptoms. The blood is of a brownish hue, but microscopically unchanged; occasionally the urine contains blood.

Measures of relief: At the first symptoms of poisoning, immediate removal from the workroom to a cool shady spot; change of clothing; cool affusions; administration of oxygen in connection with artificial respiration; in severe cases, bloodletting with subsequent infusion of physiological salt solution; copious ingestion of milk; in case of weak action of the heart, stimulants (black coffee, camphor, ether, but no alcohol); caution against the use of alcohol during and immediately after labor; abstinence is advisable.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
ANILINE DYESTUFFS: The majority of the very numerous aniline dyes are non-poisonous. Generally the basic dyes are more dangerous than the acid dyes. Regarded as suspicious or injurious to health are—			
(*a*) The various phenol nitrates, dinitrophenol, dinitrocresol (saffron yellow, aniline orange), picric acid (trinitrophenol).	Aniline dye factories; dyehouses; also manufacture of explosives.	Action on the skin; in the form of dust, through the respiratory organs; the digestive organs.	Itching, dermatitis, efflorescent eruption, yellow discoloration of the cuticle and conjunctiva; sneezing and nasal catarrh; inflammation of the buccal mucous membrane; bitter taste; disturbances of digestion; irritation of the central nervous system and of the kidneys. Picric acid is a feeble former of methemoglobin; industrial poisonings by it are extremely rare.
(*b*) The many naphthol nitrates, dinitronaphthol, Manchester yellow, dinitro and naphthol calcium; tetranitronaphthol.	Aniline dye manufactories; dyehouses.	Action on the skin; in the form of dust, through the respiratory organs; the digestive organs.	Blood poisons, forming methemoglobin. The morbid symptoms resemble those in poisoning by amido compounds; ailments of the central nervous system in great variety; paralyses.
(*c*) The nitroso dyes.	Aniline dye manufactories; dyehouses.	In the form of dust on the skin.	Intense irritation of the skin, caused, it is asserted, partly by using excessive quantities of chloride of lime in cleansing the skin.
(*d*) The aurantia—hexantrodiphenylamine; imperial yellow, its sodium salt.	Aniline dye manufactories; dyehouses.	In the form of dust on the skin.	Intense irritation of the skin, caused, it is asserted, partly by using excessive quantities of chloride of lime in cleansing the skin.
(*e*) Ethyl and methyl violet.	Aniline dye manufactories; dyehouses manufacture of colored pencils.	As dust or fine particles in the eyes.	Inflammation of the conjunctiva or the cornea.
(*f*) The Meldola dyes, corvulin, indulin, fast black.	Aniline dye manufactories; dyehouses.	As dust or atomized solution (in dyeing by the spraying process); action on the skin and respiratory organs.	Eruptions; severe irritation of the mucous membranes; uncontrollable sneezing.
(*g*) Chrysoidin, fast black.	Aniline dye manufactories; dyehouses.	In the form of dust; effect on the skin.	Eruptions (probably superinduced by the use of excessive quantities of the chloride of lime in washing the hands).
(*h*) Bismarck blue ...	Aniline dye manufactories; dyehouses.		
ANTIMONY COMPOUNDS: **Trioxide of antimony**, Sb_2O_3. **Antimony trichloride**, $SbCl_3$ (antimonious chloride, butter of antimony, antimonial ore butter). **Tartar emetic** (tartrate of antimony and potassium), $2(C_4H_4K[SbO]O_6)H_2O$. **Golden sulphide**, Sb_2S_5 (antimony pentasulphide), antimony colors.	Extraction of antimony and its compounds; burnishing of rifle barrels and steel ware; manufacture of antimony alloys, type and stereotype metal, hard lead [ammunition factories], britannia, and white metal; remelting of old and scrap metal; manufacture of aniline dyes, fireworks; vulcanizing and red-dyeing of India rubber (antimony pentasulphide); mordants and fixing materials in cotton dyeing and textile printing.	In the form of vapor (trioxide of antimony, antimonious acid, sulphide of antimony) through the organs of respiration; irritation of the skin; in the form of dust, in the manipulation of britannia and type metal.	Intensely itching eruptions of the skin, caused by local irritation and aggravated in the case of a perspiring skin; inflammation of the mouth, throat, and stomach; constipation and intestinal colic; in acute cases, diarrhea, albumin in the urine, loss of strength, weakness of the heart, vertigo, and faintness. It appears to be somewhat doubtful, however, whether all of the enumerated compounds of antimony are detrimental to the health of the workers in them.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
ARSENIC COMPOUNDS: Arsenic trioxide, As_2O_3 (arsenic, white arsenic, smelting dust); arsenous chloride, $AsCl_3$; arsenic colors, *e.g.*— **Scheele's green** (Swedish green), arsenite of copper. **Schweinfurt green** (patent, original, new, moss, mountain, parrot, May, Kaiser, Cassel, Paris, Vienna, Kirchberg, Leipsic, Würzburg, Swiss green), compound of the arsenite and the sulphide of copper. **Brunswick green,** oxychloride of copper with copper oxide and sulphate of lime. **Neuwied green.** (Similar, only a larger proportion of arsenic trioxide.) **Cochineal** (Vienna red), arsenic acid with extract of Pernambuco wood.	Arsenic mining; roasting of arsenic-bearing ores; manufacture of glass, colored chalk, chloride of arsenic for etching on brass; shot manufacture; metal working; manufacture of arsenic colors; preparation of organic dyestuffs, colored lights, textile printing, and dyeing; manufacture of wall-paper and colored paper; tanning; manufacture of oilcloth and artificial flowers; taxidermy painting (outside and decorative); pyrotechnics (Indian white-fire). It is to be observed that zinc, silver, lead, bismuth, copper, and the commercial acids often contain more or less arsenic.	In the forms of gas and dust, through the respiratory organs and mucous membranes, the stomach, and intestinal canal.	**Acute Poisoning.**—The first symptoms usually appear after half an hour or an hour, viz., constriction of the esophagus, pains in the stomach and bowels, vomiting, diarrhea, debility, cold, bluish skin, sural cramp, lowering of heart's energy, vertigo, headache, faintness, illusions, loss of consciousness, convulsions; death, sometimes choleraic symptoms. In mild cases, burning in the pharynx, vomiting, salivation, difficult deglutition and indigestion. **Chronic Poisoning.**—Constant and persistent headache combined with melancholia, disinclination to labor, and sleeplessness, which are sometimes the only symptoms; further, gastric disturbances, such as vomiting and diarrhea, which result in emaciation and decline of strength; persistent symptoms of catarrh of the mucous membranes, such as coryza, pharyngitis and bronchitis; frequently skin diseases in varying form: Erythematous, papular, and pustular cutaneous eruptions, which also produce abscesses with infiltrated and indurated borders; falling out of the hair and nails; melanosis—that is, the deposition of a brownish pigment, not containing arsenic, on the neck, trunk, and extremities.

In severe cases, disturbances of the central nervous system; intense, lightning like, lancinating pains; formication; furriness of the skin; impairment of the sensibility; chilliness; weakness of the muscles, also unilateral or bilateral paralysis, and often loss of the tendon reflexes; sometimes fever; albuminuria. The paralyses are transient, or they may last for years, leaving not infrequently permanent disturbances.

Special measures of relief: If arsenic has been ingested, thorough gastric lavage is necessary; then administer at once by the mouth five tablespoonfuls of a solution of calcined magnesia (70 g. to 500 g. of distilled water); afterward give a tablespoonful every 5 minutes until a movement of the bowels occurs; the internal use of lime water also is recommemded for rinsing out the stomach and as an antidote; to counteract the exhaustion, cold affusions, rubbing, hypodermic injections of ether and camphor.[1]

[1] Hydrated sesquioxide of iron is not mentioned.

In case of chronic arsenical poisoning: Electric vapor baths and electrical treatment are in order; the disturbances of the stomach are to be treated with calcined magnesia and unirritating liquid nourishment (milk, milk porridge, rice porridge, salep); the cachexia, by fresh air and nutritious diet, in paralyses, use iodine preparations and electricity.

ARSENIURETTED HYDROGEN, AsH_3: A colorless, extremely offensive gas with the odor of garlic.	This gas is formed everywhere when, in the use of arsenical acids and metals, hydrogen is generated for technical purposes (*e.g.*, the filling of children's toy balloons); in soldering and etching, with arsenic-containing metals or acids, *e.g.*, enamel ware factories, tin, zinc, and lead plating works; impure iron silicate, by the absorption of water, develops arseniuretted hydrogen.	In the form of a gas, through the organs of respiration (generally mixed with hydrogen).	At first no disturbance, or only slight indisposition; after some hours, chilliness, vomiting (food, bile, then blood), pain in the back, giddiness, ringing in the ears, faintness, small pulse, bluish discoloration of the mucous membranes; labored respiration; urine at times dark or even black, containing blood or hemoglobin. After 24 hours, yellow hue of the skin and mucous membranes, from absorption of biliary fluids, fetor of the mouth (resembling garlic), swelling and sensitiveness of the liver and spleen, headache, delirium, mortal anguish; death or slow convalescence.

Special measures of relief: Fresh air and oxygen; later bloodletting; use of an alkaline solution of common salt; mild alkaline drink; analeptics (coffee, camphor).

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
BENZINE: A mixture of low-ebullition portions of petroleum, known commercially under various names, *e.g.*, petroleum, benzine, ligroine, gasoline.	Benzine distillation; chemical cleansing plants, glove cleaning; removal of fat from bones, fat solvent; lacquer, varnish, and India rubber industries; manufacture of waterproof materials (application of the rubber mass dissolved in benzine); ornamental feather factories; used as a source of power.	In the form of vapor, through the respiratory organs; to a less extent, probably, through the skin also.	Headache, vertigo, nausea, vomiting, cough, irregular respiration, weakness of the heart, drowsiness, and deep sleep with cyanosis of the countenance, ...ness of the skin and complete insensibility; on awaking, headache, vertigo and depression, fibrillar twitching of the muscles, trembling, especially the musculature, as if from chills. Benzoic acid is found in the urine. **Chronic Poisoning.**—Headache, flashes before the eyes, ringing in the ears, psychosis with excitement and state resembling inebriation, sensory disturbances and hallucinations (... the prodromata of chronic benzol poisoning will also appear). The occurrence of chronic poisoning by benzine has been contested. The symptoms vary greatly because the benzine used technically is a complex mixture and not always of the same composition.

Special measures of relief: Removal of the patient into fresh air; in severe cases, stimulants, like coffee, camphor; then cold affusions.

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
BENZOL, C_6H_6: A very unstable, colorless fluid, burning with a bright, very sooty flame; extremely volatile; its homologues, *e.g.*, toluol, xylol, and cumol.	Manufacture of benzol, its homologues and numerous derivates; technical use of these products in the manufacture of colors, in carburizing illuminating and water gas; in refining and dissolving of caoutchouc, resins, fats, alkaloids, iodine, phosphorus, and sulphur; in the removal of grease from materials; dye works, laundries; lacquer and varnish factories; the rubber industry.	In the form of vapor, through the respiratory organs; reabsorption through the skin.	Benzol, its homologues and the rest of the hydrocarbons of coal tar, have a specific affinity for the central nervous system and a general action on the protoplasm of the organic cells (fatty degeneration). Female workers, particularly in their developmental years, especially at the time of menstruation, are more susceptible than men to the poisoning and in an extraordinary degree to the subacute and chronic forms of it. **Acute Poisoning.**—(*a*) *In mild cases:* Cerebral disturbances, humming in the ears, giddiness, somnolence, a condition resembling inebriation, vomiting and irritant cough, slight flushing of the face. There is often euphoria. (*b*) *In severe cases:* Symptoms on the part of the central nervous system, muscular tremor, like chilliness from exposure to cold; trembling of the whole extremities; finally, tonic and clonic spasms; euphoria; pale, livid skin; lips remarkably scarlet hued; blood bright red, thin. Discolorations of the skin like those in aniline and nitrobenzol poisoning, are wanting in benzol poisoning. (*c*) *In the most violent cases:* Hallucinations, delirium, protracted unconsciousness, and death in tonic convulsions. **Subacute and Chronic Poisoning.**—Numerous spots of extravasated blood in the skin [petechiæ] similar to those of morbus maculosus, together with severe anæmia; hemorrhage from the mucous membranes—in women from the genitals; fatty degeneration of the internal organs (heart, liver, kidneys).

Special measures of relief: Prompt removal of the patient into the fresh air; inhalation of oxygen; exclusion of female workers from every employment in which benzol is used.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CARBON DIOXIDE, CO_2: A specifically dense, odorless, colorless gas, collecting near the ground or floor.	Generated in mines by the process of breathing, by the burning of miners' lamps, and by blasting; in lime and brick kilns and dolomite calcining kilns; in decomposition and putrefaction gases; in tanneries (tan pits); in sugar mills (saturation vessels); manufacture of carbonic acid and of mineral waters; spirit distilleries, compressed yeast factories, breweries, fermenting rooms and wine cellars; in sewer and well gases; in firing and heating establishments; in the lighting of workrooms; by the exhaled air in closed workrooms and caissons.	In the form of gas by inhalation.	Large quantities occasion sudden death by suffocation. With the inhalation of smaller quantities the symptoms of illness begin with pressure in the head, vertigo, ringing in the ears and sparks before the eyes, disturbances of respiration, such as hurried breathing and pain in the chest, some times psychic excitement and convulsions. Usually in case of more protracted effect there is loss of consciousness and of the power of motion (or even death by suffocation), with gradual decline of the pulse and respiration, and often with the occurrence of delirium. On prompt removal from the poisonous atmosphere there is a restoration of consciousness with subsidence of the symptoms of illness and recovery in a few days. The occurrence of chronic poisoning by carbon dioxide is doubtful.

Special measures of relief: Examination of the air of the suspected places before entering them; immediate removal from the poisonous atmosphere; artificial respiration to be persevered in for a long time; finally inflation of the lungs with oxygen; cold affusions; stimulation of the skin; restoratives.

CARBON DISULPHIDE (carbon sulphurate), CS_2: In a pure state it is a limpid, highly refractive, extraordinarily volatile fluid, having an odor like that of chloroform; imperfectly refined, its hue is pale yellow and its odor offensive.	Manufacture of CS_2; an agent for extraction of sulphur from the mass in the process of gas purification; disinfection; a solvent for caoutchouc, gums, fats, oils, etc.; in vulcanizing caoutchouc and rubber (patent-rubber factories); for the extraction of lanolin, the refining of tallow, stearin, paraffine, and wax; production of carbon chloride; assembling and setting up carriage-wheel rims and rubber tires; imitation-silk factories.	In the form of vapor, through respiration; in fluid form, through the skin, *e.g.*, at the dipping of the hands in the fluid.	It causes heavy damage to the red blood corpuscles and to the central nervous system. **Acute Poisoning.**—In mild cases, marked stupefaction and a sense of in toxication; in more intense poisoning, pallor of the countenance, flaccidity of the arms and legs, even complete insensibility, obliteration of all reflexes, loss of consciousness, due to paralysis of the nervous system. With the inhalation of concentrated vapor there is a fatal result in a few minutes. **Chronic Poisoning.**—The earliest symptoms (first becoming manifest, sometimes after employment for a few weeks, but, for the most part, after months or even years) are headache, extending from the root of the nose to the temples, a sensation of giddiness and stupefaction, particularly at evening after the close of labor; later, pain in the extremities, muscular weakness with trembling, spasms or fibrillar twitching, also contractures, transient and permanent paralyses, with atrophy of the muscles; deafness; itching

and formication on the skin, reduction of the reflexes, circumscribed and more extensive areas of anæsthesia and analgesia; acceleration of the heart's action, nausea, vomiting, colic, alternate diarrhea and constipation, the latter condition prevailing in the later stages of the disease; emaciation, disturbance of the sense of vision, sometimes transient, but rare in the initial stage; retrobulbar neuritis, choroiditis central scotoma, disturbances of the senses of smell and taste. In respect to the central nervous system there is at first a condition of excitement, followed by depression; subsequently, very irritable, violent, and explosive temper, with hyperstimulation of the sexual instinct; later, its abnormal decline. After several weeks or months, relaxation, melancholy, a dreamy manner, weakness of memory, puerile enunciation, obtuseness.

According to Charcot, psychic disturbances occur in 87.5 per cent. of the cases. Mental diseases under the semblance of acute mania and dementia occur with good prospect of recovery; the severer forms appear in cases where there is hereditary predisposition. There have been observed also local evidences of the paralyzing effect of the carbon disulphide upon the parts brought into contact with it, especially in the fingers.

The prognosis, so far as the preservation of life is concerned, is favorable; as to the full restoration of health, it is unfavorable.

Special measures of relief: In acute poisoning, removal into the fresh air, warm baths, cold affusions; when there are symptoms of paralysis, electrical treatment; in disturbance of vision, potassium iodide and vapor baths; interdiction of the practice of dipping the unprotected hands into carbon disulphide.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CARBON MONOXIDE, CO: A colorless, tasteless gas, and, when in a state of diffusion, odorless, burning with a blue flame in the air. **Coal vapor** has from 0.5 to 5 per cent of CO. **Illuminating gas,** 6 to 10 per cent. of CO and 33 to 40 per cent. of mine gas. **Water gas,** a mixture of 41 per cent. CO, 50 per cent. hydrogen, 4 per cent. CO_2, and 5 per cent. N. **Producer gas** contains 34 per cent. CO and 60 per cent. hydrogen gas.	In industrial plants with defectively planned or ill-tended firing and heating arrangements; plants for the production of industrial gas; mining (mine gases); coal mines; blast furnaces (furnace gas); Cowper apparatus; gas purification; coke ovens, smelting furnaces; gas machines; lime and brick kilns, dolomite calcining kilns; iron and metal foundries (drying of the moulds); soldering in tin shops; charcoal burning; resin distillation; ironing; heating with open coal brasiers or coke stoves (drying the plaster and walls of new buildings); drying chambers.	In the form of gas, through the respiratory organs.	**Acute Poisoning.**—Increased blood pressure at first, with slowing of the pulse and pounding heart beat; then lowering of the pressure, with full but small pulse, and, not infrequently, with discrete spots of dilatation in the superficial blood vessels. Remarkable pale-red discoloration of the blood and of the dilated spots; formation of carbon monoxide hemoglobin is demonstrable by the spectrum.[1] (*a*) *Disturbances of the general health:* In mild cases, dull headache, flashes before the eyes, giddiness, ringing in the ears, nausea and fullness in the gastric region. (*b*) *In severe cases:* Bluish discoloration of the skin; spasmodic, wheezing respiration; sometimes tonic and clonic convulsions, more often paralytic symptoms, either with weakness of all the extremities or of the lower only, or, indeed, of only single groups of muscles, including also the facial muscles. The convulsive stage, which may be altogether absent, is succeeded by the stage of asphyxia, with sensory and motor disturbances, involuntary voiding of urine, semen, and feces; subnormal temperature; weak, slow and intermittent pulse; loss of consciousness. As sequels there have been observed pneumonias, inflammations of the skin, paralyses and psychoses, the last two often pursuing an unfavorable course. **Chronic Poisoning** (among ironers, firemen, cooks, etc.).—Frequent headaches, dizziness, nausea, vomiting, coated tongue, weakness of memory, anæmia without chlorosis; "hot flushes," formication, palpitation of the heart, insomnia, general debility and feebleness of the psychic functions.

Special measures of relief: Removal from the poisonous atmosphere; admission of fresh air; artificial respiration, with inflation of the lungs by oxygen for hours, if necessary; keep head of the injured person slightly elevated; subcutaneous injection of ether; camphor; cold affusions; rubbing; mustard poultices; electrical treatment; insufflation of ammonia vapor; administration of black coffee; alkaline salt infusion; entering where CO may be generated only when protected by safety masks and by a constant supply of air.

[1] An elementary knowledge of the function of the hemoglobin is indispensable to an understanding of the deadly effect of the transformation of hemoglobin into "carbon monoxide hemoglobin." When so changed, it is useless in the body, for it can no longer carry and distribute oxygen to the tissues. Hence all of the blood charged with this poison is virtually destroyed—lost to the system as surely as if it had escaped from a severed artery. So if a considerable proportion of the blood becomes saturated with this gas, death is inevitable, not by suffocation, as commonly imagined, but by carbon monoxide poisoning.—W. H. R.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CHLORIDE OF LIME, $CaOCl_2$: A white granular, somewhat desiccative powder, having the odor of hypochlorous acid, and containing 35 to 40 per cent. of chlorine.	Manufacture of the chloride of lime; use of the chloride of lime as an oxidizing and chlorinating agent in the chemical industry (for example, dyestuffs); disinfection; manufacture of chloroform, chlorine, oxygen; bleaching of linen, cotton, paper, cotton print works.	In the form of vapor or dust, through the respiratory organs (inhalation of chlorine gas); direct action on the skin.	More or less severe, irritating cough, symptoms or inflammation in the upper air passages; difficulty of breathing, bronchitis, asthma, sometimes hemoptysis, irritation of the conjunctiva, lachrymation; skin hot from action of chlorine; hyperhidrosis; intensely itching and burning eruption on the skin, eczema, burns from the dust of lime and its chloride.

Special measures of relief: Admission to the employment of such, and only such, workmen as are sound and strong and free from any predisposition to catarrhal affections; technical arrangements which permit the charging and emptying of the chambers from the outside.

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CHLORINE, Cl: A yellowish-green, suffocating gas, of penetrating odor, which forms a solution of a greenish-yellow color when dissolved in water.	Manufacture of chlorine, chloride of lime, and of organic chlorine products; bleacheries; paper mills; laundries; ironing; tinning works; manufacture and use of disinfecting agents containing chlorine.	In the form of gas, through the respiratory organs.	The smallest quantities excite severe suffocative sensations and necessitate leaving the room, so that acute chlorine poisoning seldom occurs. **Symptoms of Cutaneous Disease.**—Burning, stinging, formation of nodules, blebs, and even open wounds of the skin. **Effect on the Mucous Membranes.**—Lachrymation, coryza, cough, oppression of the chest and intense dyspnœa; bronchial catarrh with hemorrhage; sometimes lobular pneumonia. The concentrated vapor causes uncontrollable cough, spasm of the glottis, dyspnœa, cold sweats, cyanosis and small pulse; death occurs within a few minutes (sudden collapse). **In Its Chronic Effect.**—Distress in the gastric region; chronic catarrh of the stomach; pyrosis; pallid countenance; catarrh of the respiratory tract; lobular pneumonia; headache, vertigo, insomnia; gradual emaciation and premature senescence. **Chlorine Acne.**—(Occasioned in the electrolytic production of chlorine by chlorinated carburetted hydrogen.) Inflammatory processes in the dermal glands; the occurrence of unusually diffuse, confluent comedones with indurated, dark-green heads; solid infiltration of the sebaceous follicles, their inflammation and suppuration causing pustules and boils.

Special measures of relief: Removal of the patient into the fresh air; inhalation of amyl nitrite; artificial respiration; on account of the paralyzing effect of the chlorine on the heart, stimulants are required (black coffee, subcutaneous injection of camphorated oil); to control the irritating cough, hypodermics of morphine or cautious inhalation of steam.

For the prevention of chlorine acne: Substitution of anodes made of molten metallic oxides for the carbon anodes.

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CHLORODINITROBENZOL, $C_6H_3(NO_2)_2Cl$: Forming yellow crystals. (*See* Nitrobenzol.)			
CHLORONITROBENZOL, $C_6H_4NO_2Cl$: Forming yellowish crystals of aromatic odor. (*See* Nitrobenzol.)			

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CHROMIUM COMPOUNDS: Chromic acid, anhydrous, CrO_3; chromates and bichromates, *e.g.*, sodium chromate, Na_2CrO_4; sodium bichromate, $Na_2Cr_2O_7$; lead chromate, $PbCrO_4$. **Chromium colors:** Chrome yellow (acid chromate of lead); chrome orange (basic and neutral chromate of lead); chrome red (chrome-cinnabar); acid chromate of lead oxide and lead hydrate; chrome green, poisonous only as a mixture of chrome yellow and paris blue. (*See also under* Lead.)	Manufacture of chromium preparations, chrome colors, and hectograph composition; photography (color and carbon printing); oxidizing agent in the tar-color industry; manufacture of matches; wet batteries; bleaching fats, oils, and wax; mordant in Turkish red dyeing, textile printing (for neutralizing colors and for dyeing); chrome tanning (two-vat process); staining of wood.	Absorption by the skin and mucous membranes; in the form of dust, through the respiratory organs.	The chromates act very much like chromic acid itself; pit-like, phagedenic ulcers, burrowing deep and spreading wide, very difficult to heal and very painful, occur almost exclusively on the skin of the hands, more rarely on the arms, thighs, scrotum, and penis, resembling syphilitic ulcers; they also appear, though seldom, on the mucous membrane of the tonsils and of the hard and the soft palate. With rare exceptions is there extension of the inflammation to, and perforation of, the nasal septum at the cartilaginous portion; eczematous eruptions. Irritation of the conjunctiva. Irritation of the Bronchioles.—Chronic bronchial catarrh, and small areas of inflammation in the lungs. In recent years the last-mentioned symptoms are hardly ever encountered in a remarkably wide field of observation. It is at least extremely doubtful if disease of the kidneys is ever caused by chromium. In handling chromium dyes containing lead there is danger of chronic lead poisoning.

Special measures of relief: Chromium ulcers are successfully overcome by careful treatment of the slightest injuries to the skin, and by the immediate, complete, and skillful closure of the lesions.

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
CYANOGEN COMPOUNDS: Dicyanogen, C_2N_2; prussic acid, HCN: Hydrocyanic acid, a colorless, highly volatile fluid, of penetrating, pungent, and irritating odor. **Natrium cyanide** (NaCN), Cyanide of potassium, potassium cyanide, (KCN): A colorless salt, forming crystals which, after fusion, recrystallize, but readily decomposes on exposure to the air, setting free hydrocyanic acid. **Rhodanic** (sulphocyanic, SCN) **compounds:** Poisonous dose of the dilute hydrocyanic acid, 0.06 g.	Extraction of gold; silver and gold plating, galvanoplasty, electroplating; manufacture of cyanogen compounds and inorganic processes (when organic residue are heated with alkalies); reduction of residuum to gas; blast furnaces; gas works (purification process), dye works and printeries; photographic establishments; manufacture of celluloid.	In the form of gas, through the respiratory organs; prussic acid also through the epidermis.	Generally speaking, industrial poisonings by cyanogen are rare. **Acute Poisoning.**—Moderate quantities of the gas cause vertigo, headache, rush of blood to the head, oppression of the chest, palpitation of the heart, a sensation of constriction at the throat with pharyngeal irritation and dryness, nausea and vomiting, difficult, gasping respiration, with retention of consciousness. To the stage of dyspnœa succeeds that of spasm with cold, perspiring skin, convulsions and involuntary micturition, with loss of consciousness. In the stage of asphyxiation there are temporary suspension of respiration, retardation of the heart's action, lividity of the skin and mucous membranes, lowering of the body temperature; with inhalation of large quantities, the stage of asphyxia supervenes immediately. Dilatation of the pupils; loss of consciousness; a few gasping inspirations; cyanosis of the skin and mucous membranes; collapse; death. **Chronic Poisoning** (very doubtful).—Headache, vertigo, unsteadiness of gait; nausea, loss of appetite, disturbances of the gastric and intestinal functions; slowing of the pulse; albuminuria.

Special measures of relief: Fresh air; artificial respiration; administration of oxygen; cold affusions and friction; hypodermatic injection of ether, camphor; if the poison has been taken into the stomach, give emetics, then immediately rinse out that viscus with water, with the addition of one-quarter to one-half of 1 per cent. of potassium permanganate. Kobert recommends a 3 per cent. solution of hydrogen binoxide for subcutaneous injection, in doses of 1 cc. at different points in the body. But on the other hand, H_2O_2 is deemed unsuitable, and an alkaline solution of ferric sulphate or an antidote for arsenic with some ferric salt is indicated as the best remedy. To control the convulsions give morphia hypodermically.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
DIAZOMETHANE, CH_2NH_2: A very volatile yellow gas.	In methylizing of every kind.	As gas, through the lungs; effect on the skin.	**Acute Poisoning.**—Severe headache; great physical depression; grave lesions of the lungs; other effects like those of dimethyl sulphate.
DIMETHYL SULPHATE, $(CH_3)_2SO_4$: A colorless oily fluid.	Production of methyl ethers, methyl esters and methyl amines; manufacture of artificial perfumes.	In the form of gas, through the respiratory organs; direct action on the skin.	Strongly corrosive effect on the skin and mucous membranes; burns; pains in the nape of the neck and in the thoracic cavity; hoarseness; destruction of the mucous membrane and aspiration of the broken-down products into the lungs; lachrymation, conjunctivitis, formation of erosion-eschars, and œdema, photophobia and parenchymatous clouding of the cornea; even coma, convulsions, paralysis, and a fatal outcome.
DINITROBENZOL or **BINITROBENZOL,** $C_6H_4(NO_2)_2$: When pure, crystallizing as slender, colorless, rhombic needles; when impure, in yellow, crystalline cakes. (*See* Nitrobenzol.)			
FORMALDEHYDE, CH_2O: A liquid, volatilizing as a gaseous vapor of penetrating odor; 10 per cent. formaldehyde, formalin.	Disinfection; manufacture of many organic preparations, especially in the coal-tar color-industry; preserving and hardening of human and zoological preparations.	In the form of vapor, through the respiratory organs and mucous membranes.	Intense irritation of the skin and mucous membranes.

Special measures of relief: Do not enter the disinfection chamber until after the introduction of ammonia and thorough ventilation.

HYDROCHLORIC ACID, HCl: Pure HCl is a colorless gas that fumes when open to the air, forming a dense, acid, white mist. The crude commercial hydrochloric acid is, for the most part, impure, containing arsenic among other admixtures.	Treatment with chlorine of previously roasted ores; potteries (glazing), enameling works, glass factories, soldering; in the chemical industry, manufacture of chloride and sulphate of soda, of muriatic acid, stannic acetate, etc.; manufacture of artificial fertilizers; bleaching, shoddy industry; cotton-print works; carbonizing of materials; India-rubber industry.	Action on the skin and nasal mucous membrane; seldom in vaporous form, affecting the respiratory organs.	As a rule the rarefaction of the hydrochloric acid gas is so considerable in the industries where it is used to any extent worth mentioning that only in exceptional cases do injurious effects occur, such as irritation of the respiratory organs. A proportion of 0.05 per mille of hydrochloric acid in the air is well borne, but only for a short time. A greater concentration (as well as the often-repeated inhalation even of moderate quantities in manufacturing industries) causes chronic irritation of the mucous membranes to which the vapor has access. There result also catarrh of the conjunctiva, coryza, pharyngeal, laryngeal, and bronchial catarrh, together with dental caries. Concentrated HCl vapor may cause unconsciousness and death.

Special measures of relief: Removal of the patient from the dangerous atmosphere; inhalation of a finely nebulized solution of sodium bicarbonate.[1]

[1] In addition, for acute poisoning, give atropine (1/60 grain) subcutaneously to stimulate the pneumogastric.—W. H. R.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
HYDROFLUORIC ACID or FLUORIC ACID, HF: A colorless gas, of pungent odor and forming a dense mist in the air.	Production in chemical works; glass factories, etching on glass; laboratories of the pottery industry; extraction of the fluorides of antimony (substitute for tartar emetic in dye works); fertilizer factories (extraction of phosphorites) bleaching of cane for chair seats and extraction of its silicates.	In the form of gas, through the respiratory organs. In a fluid state it has an immediate action on the skin and mucous membranes.	Intense irritation of the eyelids and conjunctiva, coryza, bronchial catarrh with spasmodic cough, ulceration of the nostrils, gums, and oral mucous membrane; also painful ulcers of the cuticle, erosions and formation of vesicles; suppuration under the finger nails.
LEAD, Pb: A bluish-white, highly lustrous metal, which on exposure to the air acquires a gray tarnish. **Lead alloys.** **Lead colors;** other lead compounds. **Lead Sulphuret** (galena) is held to be non-poisonous, and some lead polysilicates are regarded as nearly so.	Smelting of lead and lead-bearing ores; manufacture and use of articles made of metallic lead (sheets, plates, boxes, pipes, wire, cans, flasks, pails, kettles, faucets, retorts); manufacture and use of lead alloys, as type metal, shot (tin foil), for example, in type foundries, tin shops, bottle-cap factories, composing rooms, file-cutting works; manufacture and use of lead colors and other lead compounds, as litharge, white lead, Krems white, red lead, lead chromates, acetate of lead, lead chloride in lead color works and storage-battery factories, in the trade of painter, house painter and varnisher; plants for installation of gas and water; in the ceramic industry, the textile industry, etc. It is to be observed that materials containing lead may occasionally be employed in every industry, and that lead colors and other lead compounds are often met with in trade under fanciful names.	Absorption of lead and lead compounds occurs: (1) In isolated cases through the skin; whether through the uninjured skin is doubtful; (2) in the form of vapor (very finely divided oxide of lead), and as dust, through the respiratory organs; (3) by way of the digestive tract by means of contaminated food and drinks (for example, cigars, cigarettes, chewing tobacco). By inhalation the dust, laden with lead, finds lodgment in the upper respiratory tract, and, mixed with saliva, may reach the stomach.	Industrial lead poisoning appears as a rule in the chronic form and arises from continuous absorption of the most infinitesimal quantities of lead during a protracted period of time (weeks, months, and even years). The beginning is insidious, with disturbances of the general health, a sense of weakness, decline of bodily strength; sallow, pale-yellowish hue of the skin. Distress in the region of the stomach, eructations, lack of appetite, metallic taste in the mouth and fetid breath. The blue line (blue-gray discoloration of the gums) which, however, may be absent, even in the course of a severe attack; lead colic with most obstinate constipation, retention of urine; plumbic arthralgia (lacerating, boring) occurring for the most part paroxysmally, chiefly in the lower extremities, more rarely in the upper, often interpreted as a symptom of rheumatism of the joints; frequently, fibrillar trembling of the fingers. Typical are the lead paralyses, of which disturbances of sensation (paræsthesia and anæsthesia) take the precedence. Paralysis generally affects the extensor muscles of the arm and hand, with atrophic manifestations; more rarely, the flexor muscles. Sometimes also there are paralyses of the estensors and flexors of the lower extremities or muscles of the shoulder. From experience it is known that those groups of muscles are especially affected which are most used in the occupational activity. Transient blindness, but also gradually progressive atrophy of the optic nerve; temporary loss of the special senses of smell and taste; violent, often fatally ending disease of the brain (saturnine encephalopathy), sometimes preceded only by slight premonitory symptoms, as irritability and headache, ringing in the ears, insomnia; more often, slowly increasing mental disturbances precede; epileptiform convulsions, hallucinations; morbid changes in the blood vessels and of the heart and kidneys (contracted kidney); increase of blood pressure and granular degeneration of the red blood corpuscles. Disturbances in the sexual sphere in women; abortion, premature birth, low vitality of the children.

Measures of relief: Discontinuance of work in lead at the slightest symptoms of lead poisoning. In lead colic, give first, by the mouth or subcutaneously, morphia, opium, or atropine; afterward, cathartics (castor oil or podophyllin); in paralysis, electrical treatment, massage and baths; in every case, strengthening diet, iodide of potassium, and sudorifics.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
MANGANESE DIOXIDE, MnO_2: Brown mineral (occurring chiefly as pyrolusite).	Breaking and grinding of manganese ore; sifting out of the refuse.	In the form of dust, through the respiratory organs.	MnO_2 produces cumulative effects. After protracted action of the agent the symptoms begin with disturbances of the general sensibility, general debility, languor, lancinating pains in the extremities, in the small of the back and nape of the neck, creeping sensations in the legs and numbness in the feet; salivation; tremor of the head, tongue, and hands; later, locomotor disturbances with uncertain, stamping gait, and, ultimately, the impossibility of safe and sure progression. Affections of the voice (low, whispering) and of speech (indistinct, scanning) combined with flatness of tone; forced laughter and weeping and lowering of intelligence. Sometimes dropsical effusion into the cellular tissue of the lower extremities.
MERCURY, Hg: A silver-white, shining metal, unchangeable in the air, but evaporating at house temperature. **Mercury compounds,** amalgams (alloys with metals). **Cinnabar** (HgS) is non-poisonous.	Mining and smelting of quicksilver; occupation of mirror plater, amalgam gilding and silvering; manufacture of thermometers, barometers, and manometers, incandescent electric lamps, Röntgen and Hittorf tubes, mercurial vapor lamps; manufacture of the salts of mercury, amalgams, and colors, pharmaceutic products, antiseptic dyes, inflammable materials, and explosives; employment of the salts of mercury, especially in the hare's fur business and felt-hat manufacture; photography and steel engraving.	Absorption through the uninjured skin; absorbed in the form of vapor and as dust (amalgam dust, dust of the compounds of mercury).	Industrial mercurial poisoning is a chronic poisoning occasioned by work in this metal for a long period, commonly weeks, months, years, or decades. The first symptom is generally increased ptyalism, with swelling and inflammation of the gums and of the buccal mucous membrane, often with the formation of rodent ulcers, besides, there are, frequently, disturbances of digestion, lassitude, and pallor. Associated with the further absorption of mercury, "erethism" supervenes—a peculiar psychic excitability (timorousness, bewilderment, irritability) aside from the characteristic mercurial tremor. In a state of complete repose this tremor is not noticeable, and manifests itself only on voluntary movement, causing a quite distinctive, irregular tremulousness of the fingers, hands, arms, and finally, also, of the legs and head. In strictly chronic cases the stomatitis and erethism are absent, and only the tremor is observable. Death may result in the worst cases in consequence of the violent tremor and spasms affecting the entire body; in other cases, increasing weakness. Cachexia.

Special measures of relief: Relinquishment of the employment; nutritious diet; vapor baths; potassium iodide.

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
METHYL ALCOHOL (wood spirit), CH_3OH: A colorless fluid of faint odor.	Produced by the dry distillation of wood; used in the preparation of varnish, lacquer, polish, and perfumes; for the denaturing of spirits; for the production of coal-tar colors and pharmaceutical preparations; a solvent for aniline dyes in cotton-print manufacture; used in combination with shellac for coating the interior of casks; in cabinet making, furniture polishing and paint removing.	Absorption through the digestive organs, also through the skin; in the form of vapor, through the organs of respiration.	The effect is very persistent; nausea, headache, ringing in the ears, weakness of the muscles, insomnia, delirium, difficulty of breathing, and sometimes deafness; inflammation of the throat and the mucous membrane of the air passages extending to the finest ramifications of the bronchial tubes; finally, death by paralysis of the respiratory apparatus. Conjunctivitis; also serious affections of the retina and the optic nerve, resulting in blindness, even, from atrophy of this nerve.[1] In chronic cases, fatty degeneration of the liver and acidosis.[2]

Special measures of relief: The substitution of innocuous media for methyl alcohol in the denaturing of spirits.

[1] Permanent blindness and even a fatal issue may be caused by the ingestion of small quantities of wood spirit; hence the risk incurred in using cheap essences of vanilla and other flavoring extracts which contain methyl alcohol.—W. H. R.

[2] J. A. M. A., Oct. 25, 1913, page 1544.

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List of Industrial Poisons—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
METHYL BROMIDE, CH_3Br: A colorless, gaseous body of aromatic odor. **Methyl iodide, iodine methylate,** CH_3I: An ethereal, colorless fluid of somewhat penetrating odor, soon becoming yellow on exposure to the air.	Employed in aniline dye factories.	In the form of gas, through the respiratory organs and the mucous membranes.	In mild cases, vertigo, headache, and transient stupor, with diplopia and a sensation of rigidity in the muscles of the eyes. In a severe case there was observed loss of consciousness continuing 8 weeks, with staring look, pallor of the skin, retarded pulse, and obstinate constipation. During brief intervals of wakefulness there was unrest with increasing excitability. (Grandhomme.)
NITRANILINE, $C_6H_4NH_2NO_2$: Forming long, yellow crystals. (*See* Aniline.)			
NITROBENZOL (mirbane oil, imitation bitter-almond oil), $C_6H_5NO_2$: A colorless, highly refractive fluid having an odor like that of bitter almonds; and all nitro-compounds of benzol and its homologues, *e.g.*, dinitrobenzol, dinitrochlorobenzol, nitrotoluol, nitrophenol, nitronaphthalene, etc. The most of the nitro- and chloro-compounds are the more poisonous.	Coal-tar color industry and those establishments in which its intermediate products are manufactured, as in explosives works, perfumery and soap factories, pharmaceutical laboratories, etc.	(1) Absorption takes place, first of all, through the skin, both the uninjured and especially the pathologically altered skin, particularly in the case of profuse perspiration; (2) through the respiratory organs; (3) through the digestive organs.	Poisoning by all of the designated substances is pretty nearly the same, qualitatively; quantitatively, however, differences exist, so that the larger proportion they contain of the nitro (NO_2) groups the more virulent they are likely to be. The nitrochloro-compounds are very much more dangerous than the simple nitro-compounds. The first toxic symptoms may appear within a few hours (8 to 24) after absorption of the poison. **Acute Poisoning.**—(*a*) *In mild cases:* Malaise, headache, giddiness, nausea, loss of appetite, costiveness, burning sensation of the skin and mucous membrane. (*b*) *In severe cases:* A feeling of anxiety, disturbances of sensation, like formication on the legs and furriness of the soles of the feet, ringing in the ears; disturbances of coördination (reeling gait, stammering speech), increased excitability of the reflexes, convulsions and a state of general spasm; later, with decline of sensibility, symptoms of paralysis; vomiting; odor of the vomitus and of the exhaled breath like that of bitter-almond oil; icterus of the skin; at first increased, afterward diminished activity of the heart, with lowered tension of the pulse; visual derangements (amblyopia, optic neuritis); blood viscid, brown to deep dun color; diminution of the red corpuscles and alterations in their form; in the advanced cases, formation of methemoglobin. The course of severe cases is exceptionally varied; after intermissions, exacerbations may occur with a finally fatal result. Death may occur also in connection with deep insensibility, without other symptoms. The symptoms which point to blood changes predominate, in severe poisoning, over the nervous symptoms. **Subacute and Chronic Poisoning.**—Icterical skin, which gradually becomes cyanotic, methemoglobin formation; symptoms of degeneration and regeneration of the red-blood corpuscles; general debility, anæmia. The clinical picture is similar to that of pernicious anæmia. In the urine the poisoned corpuscles are sometimes demonstrable, and finally the presence of hematophorphyrin and of albumen.

Measures of relief: Immediate removal from the workroom; inhalation of oxygen; artificial respiration; eventually bloodletting; stimulants, non-alcoholic; prohibition of the use of alcoholic drinks during working hours; avoidance of the same, also, outside of employment.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
NITROGYLCERINE, $C_3H_5O_3(NO_2)_3$, glycerine trinitrate: An oily, vaporable, colorless fluid, without odor.	Manufacture of explosives (dynamite, nitro-cellulose); in the use of dynamite.	Inhalation of the vapor; absorption through the uninjured skin, mucous membranes, and wounds of the skin. In the explosion of dynamite the action of carbon dioxide and nitrous monoxide, as well as that of undecomposed nitroglycerine is present.	Extraordinary toxicity, somewhat like effects of prussic acid; just a few drops are deadly, and even mere contact with products containing nitroglycerine may cause poisoning; severe headache, disturbance of the intellect, facile syncope, vertigo, burning in the throat and stomach; nausea; vomiting, colic; symptoms of paraylsis in the muscles of the head and eyes, as well as in the lower extremities; bradycardia and retarded respiration, stertorous breathing and dyspnœa; cyanosis; coldness of the extremities; injection of the conjunctiva; reddening of the countenance. *In the mixing and sifting of dynamite:* Obstinate ulcers under the nails and on the finger tips, eruption on the plantar aspect of the feet and interdigital spaces of both hands, with extreme dryness and formation of fissures. *Explosion of nitroglycerine with little gas:* Trembling, determination of blood to the head, vomiting, headache. *Explosion of nitroglycerine with much gas:* Vertigo, asphyxia, cyanosis, motor paralysis and loss of consciousness; intermittent, stertorous respiration, coldness of the skin, small pulse; after recovery of consciousness, debility, nausea, vomiting, headache, intermittent pulse, and finally death. Chronic Poisoning.—Disturbances of digestion, trembling, neuralgia.
Special measures of relief: Absolute avoidance of contact.			
NITRONAPHTHALENE, $C_{10}H_7(NO_2)$: A yellow, friable, crystalline mass of strongly aromatic odor. (*See* Nitrobenzol.)			

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List of Industrial Poisons—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
NITROUS GASES (low degrees of oxidation of nitrogen, which appear simultaneously): Nitrogen protoxide, NO; nitrogen deutoxide, NO_2; nitrogen trioxide, N_2O_3; anhydrous nitrous acid (HNO_2). Red fuming nitric acid is a saturated solution of N_2O_4 in crude NHO_3. NO is a colorless gas which under the influence of atmospheric oxygen is readily transformed into brown nitrogen dioxide. Below $-20°C$. N_2O_3 is a blue fluid; at the ordinary temperature it separates into NO and NO_2.	Nitrous gases are produced by the action of nitric acid on deoxidating substances of various kinds, principally on metals (iron, lead, zinc, etc.), on organic substances (coal dust, wood, straw, paper, textile fabrics, woollen refuse, etc.) as well as many other substances (pyrites, sulphurous acid and its salts, soda sediment, hydrocholoric acid, iron chlorides, sulphate of iron, etc.); in the preparation of nitric acid, its combinations and salts, among which the nitrous salts also are to be included; metal etching and metal refining; stamp mills and mints; galvanotechnics; nitrification in chemical works and manufactories of explosives; celluloid manufacture; sulphuric acid manufacture; production of picric acid, aniline colors, nitrocellulose (gun cotton, collodion cotton), xyloidine, nitro-starch, nitrojute dynamite, abelite, nitromannite, nitrosaccharose, viscosine, etc.; nitric acid manufacture and storage; preparations of thorium and cerium; bleaching materials (oils, etc.); hat making (maceration of the hair); etching and engraving on copper (etching of the plate); dyeing and printing (fixer and mordant).	In gaseous form, through the respiratory organs.	Susceptibility to the effects of nitrous gases fluctuates considerably. Persons who suffer from diseases of the respiratory organs are especially susceptible; not infrequently the continual inhalation of small quantities, for many consecutive years even, occasions no serious disturbances of the health. A pale, sallow complexion and chronic bronchial catarrh may be deemed, nevertheless, the usual consequences of occupational inhalation of very moderate quantities of nitrous gases. Often, however, larger quantities of the poisonous gases are borne for hours together (6 to 8 hours) without discomfort; when suddenly, after a long interval without disturbance, ominous symptoms appear. Symptoms of irritation in the air passages are manifest, as a feeling of constriction of the larynx, spasmodic cough, oppression in the chest, labored respiration, anxiety, cold perspiration on the face, protrusion of the eyes, gasping speech, paroxysms of coughing, bluish discoloration of the countenance, coldness of the extremities. Consciousness is at first unimpaired, but with increasing difficulty of breathing it becomes dimmed; injury to the teeth. The urine is scanty, brown in color, containing hemoglobin and albumen. Death results from œdema of the lungs. In very severe cases methemoglobin is observed, and then a general systemic poisoning may result.

Special measures of relief: Immediate removal from the noxious atmosphere; inhalation of oxygen; finally, bloodletting and infusion of normal salt solution.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
OXALIC ACID, $C_2H_2O_4$: It forms large, pellucid crystals.	Manufacture of oxalic acid; polishing of metals, especially of copper and brass utensils; used in dye works, chemical cleansing plants (rust and ink stains); straw hat manufacture and straw braiding.	In the form of dust, through the respiratory organs.	Opalescent or bluish discolorations (with brittleness) of the nails; blood stasis in the hands; corrosive action on the mucous membrane of the œsophagus, of the stomach and bowel; weakness of the heart; convulsions and spasms. However, industrial poisonings by oxalic acid are exceedingly rare.
PETROLEUM: A mixture of various hydrocarbons of the methane, ethyl, and aromatic series.	Production of oil; refining of the crude oil; furniture polishing by use of so-called polishing oil.	In the form of vapor, through the respiratory organs. As a fluid it has a direct action on the skin.	The vapors of petroleum cause a profound acute poisoning with a condition of inebriation; shouting, reeling, and prolonged sleep without any recollection of what has happened; in severe cases, loss of consciousness, lividity of the countenance, staring look and contracted pupils, almost imperceptible pulse, asphyxia. The chronic effect of petroleum vapor causes numbness and irritation of the Schneiderian membrane. In general, the symptoms of the action of petroleum resemble those resulting from the action of benzine. By reason of the high boiling point of petroleum there are produced, in the extraction of paraffine butter, in the emptying of retorts, and in the filling of casks with petroleum, obstinate inflammations of the hand in the form of acne (nodules, pustules, and boils).

Special measures of relief: Removal into the fresh air; in collapse, a tepid bath with cold affusions; subcutaneous injections of camphorated oil.

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
PHENOL, C_6H_5OH (carbolic acid): A white crystalline mass, and its homologues, *e.g.*, cresol, lysol, and their derivatives.	Anthracite coal tar distillation; production of picric acid and of many organic aromatic compounds; used in dyeing, calico printing; manufacture of lampblack, in photogen factories; impregnating wood with tar and oil of tar; surgical dressing industry.	Action on the epidermis and the digestive tract.	Erosion of the skin, which by great extension may lead to severe internal injuries; symptoms of degeneration in the blood and in the internal organs (nephritis); gangrene, icterus, collapse.
PHENYLHYDRAZINE, $C_6H_5NH{\cdot}NH_2$: A yellowish, oily fluid, shading into brown, of pungent color.	A by-product in the manufacture of antipyrine from aniline; manufacture of organic compounds.	Absorption by the skin; action on the skin.	Obstinate vesicular eruption on the skin, with itching and burning; diarrhea, loss of appetite; granular degeneration of the blood corpuscles; formation of methemoglobin; a sense of general malaise.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
PHOSGENE, $COCl_2$ (carbon oxychloride): A colorless gas, of suffocating odor.	In the manufacture of phosgene and its use for the production of organic compounds.	In the form of vapor, through the respiratory organs.	Until the present time only the acute form of poisoning has been recognized. The first symptoms of illness sometimes appear only after many hours. By means of the hydrochloric acid arising from the decomposition of the gases in the lungs, destruction of lung tissue results, with difficulty of breathing, paralysis of the lungs, and pulmonary oedema. A fatal outcome is often observed.

Special measures of relief: Inhalation of oxygen and medical attendance immediately after breathing the phosgene gas.

PHOSPHORUS, P: A colorless, transparent substance; on exposure to the light, translucent and of a yellowish, waxy luster. In the air it is luminous, and when heated in closed iron crucibles to a temperature ranging from 250° to 300°C. it is converted into red or amorphous phosphorus, which is unaffected by the air. The yellow or white phosphorus is very poisonous; the red, non-poisonous.	Extraction of phosphorus from phosphorites and coprolites, bone-black (refuse of sugar mills), bone-ash (refuse of meat extract manufacture); production of phosphor-bronze, of phosphorus compounds, igniting agents, matches, and tar colors.	In the form of vapor, through the respiratory organs; into the digestive canal by means of food contaminated by the fingers; action on the skin.	As industrial poisoning it occurs only in the chronic form, occasioned by the absorption of very minute particles of the poison for a period of months, generally, indeed, of years. Symptoms of the disease sometimes first appear long after relinquishment of the occupation. It is doubtful whether chronic phosphorism occurs (that is, general systematic poisoning by phosphorus). Chronic phosphorus poisoning uniformly affects the bones of the face, beginning with inflammation and sclerosis of the bones and of the periosteum; then, by extension of the suppurative process, necrosis results. This most frequently attacks that portion of the alveolar process of the jawbone which is least protected against infection. Swelling and ulcerations on the gums and the buccal mucous membrane, pain even in the sound teeth, loosening and falling out of the teeth, infiltration of board-like hardness occurs in the soft parts surrounding the jaw; suppuration and destruction of the jawbone (necrosis) with numerous fistulous channels which here and there burrow through the cheek. Hand in hand with the ulcerative processes go osteoplastic formations, so that, while suppurative destruction of tissue takes place at one point, at another the formation of new bone is going on. The under jaw is more often affected than the upper; here the process goes on insidiously without formation of new bone but with local destruction of the part. The palatal and orbital bones may be attacked with ulceration and shrinking of the eyeball. By extension of the inflammation along the sheaths of the vessels there result meningeal inflammation and cerebral abscess. There is remarkable brittleness of the bones, decline of appetite, pallid complexion, diarrhea, emaciation. Sometimes there is amyloid degeneration of the abdominal organs. Death by sepsis.

Special measures of relief: To the utmost possible extent the prohibition of the use of white or yellow phosphorus; exclusion of laborers that have dental caries, after extraction of a tooth at least 2 weeks' exclusion from the employment; change of occupation; improvement of the general health; there is no specific medical treatment; in appropriate cases, operative intervention.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
PHOSPHORUS SESQUISULPHIDE, P_4S_3: A grayish yellow, odorless and tasteless substance.	In chemical factories.	Inhalation of sulphuretted hydrogen in the fusion of phosphorus and sulphur as well as in the drawing off of the molten mass from the kettles; dust in grinding and sifting of the paste; bicarburet of sulphur vapors in the extraction of yellow phosphorus and regeneration of CS_2.	Irritation of the mucous membranes, especially obstinate conjunctivitis. Through the influence of dust in the grinding and sifting of the composition there appear symptoms of CS_2 poisoning. To be noticed also is the danger of poisoning by sulphuretted hydrogen. (*See under* Sulphuretted hydrogen.)

Special measures of relief: Prevention of the contamination of phosphorus sesquisulphide with yellow phosphorus; precautions against injury from the effects of sulphuretted hydrogen.

PHOSPHURETTED HYDROGEN, PH_3: A colorless gas of nauseating odor.	In the extraction of phosphorus; in the preparation of red phosphorus and the sesquisulphide of phosphorus; in the reduction of iron silicate containing phosphorus by the action of moisture; in the production of acetylene with calcium carbide that contains an admixture of calcium phosphate.	In the form of gas, through the respiratory organs.	An anxious, oppressed feeling in the chest, changing to a burning, lancinating pain; affections of the head, vertigo, tinnitus aurium; general debility; loss of appetite; great thirst. Death occurs without convulsions, through the effect of the poison on the blood.
PICRIC ACID, $C_6H_2(OH)(NO_2)_3$: Trinitrophenol in a pure state forms pale-yellow, bitter tasting, foliate, metallic crystals.	Chemical works, dye-houses; manufacture of explosives and powder (lyddite, melinite); projectile factories, filling shops.	In the form of dust, through the respiratory passages; direct action on the skin.	Poisonings with picric acid are rare; when they occur there are itching; inflammation of the skin, vesicular eruption, yellow pigmentation of the epidermis and of the conjunctiva, inflammation of the buccal mucous membrane, bitter taste, disturbances of digestion, epigastric pain, nausea, vertigo, diarrhea, and jaundice; picric acid decomposes the constituents of the blood. By the penetration of dust into the nostrils, sneezing and nasal catarrh are occasioned.
PYRIDINE, C_5H_5N: A colorless fluid of pungent and characteristic odor. Its homologues, pyridine bases.	In its manufacture out of coal tar and bone tar; in the use of denaturing spirits (shops for wood-working, gilding, and hat manufacture).	In the form of vapor, through the respiratory organs. In a fluid state it acts on the skin of the hands and arms.	Catarrh of the mucous membranes; hoarseness, irritation, and choking sensation in the throat; headache, vertigo, flaccidity and trembling of the extremities; difficulty of breathing and clonic convulsions; eczema of the hands. Industrial poisoning by pyridine is very rare.
SULPHUR CHLORIDE, S_2Cl_2: A thickish fluid, of brownish color and suffocating odor, fuming on exposure to the air.	Solvent for sulphur and fats; caoutchouc and patent rubber industry.	In the form of vapor, through the respiratory organs.	In contact with water and atmospheric moisture, it is resolved into hydrochloric acid vapor. The vapor of sulphur chloride is suffocating; if ingested, it excites vomiting.

Special measures of relief: Wearing of rubber gloves; instant removal of the patient from the poisonous atmosphere.

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LIST OF INDUSTRIAL POISONS—*Continued*

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
SULPHUR DIOXIDE, SULPHUROUS ACID, (H_2SO_3): Its anhydride is SO_2, in the form of gas; condensed, it becomes fluid. The gas is of pungent odor and suffocating effect.	Roasting of sulphur-bearing ores; brick works, ceramic industry; manufacture of sulphuric acid, of ultramarine; extraction of bones, manufacture of glue and gelatine from bones; disinfection; refining of petroleum; manufacture of candles; bleaching of wax, silk, and wool; chromium tanning two-vat process); bleaching of straw hats and bristles; preserving wine and fruits; fumigating hops and casks with sulphur; ice machines; heating plants (burning of pyrite-bearing coal.	In the form of gas, through the respiratory organs.	In moderate concentration sulphurous acid is borne without inconvenience or injury; persons accustomed to the gas bear very well a proportion of 0.003 to 0.004 per cent. of SO_2 in the air. Susceptible persons, at the beginning of their employment in an atmosphere containing sulphurous acid, manifest a transient irritation of the mucous membrane of the respiratory organs and of the eyes. In its severe action there is spasmodic cough with secretion of tenacious, often blood-tinged, mucus. The protracted effect of a high degree of concentration is livid discoloration of the mucous membranes, bronchial catarrh, croupous angina of the bronchi and their branches, and inflammatory areas in the lungs; disturbances of digestion.

Special measures of relief: Removal from the noxious atmosphere; admission of fresh air; artificial respiration; infusion of weak alkaline solutions (0.05 to 0.1 per cent. liquor natrii caustici [*solution of caustic soda*]).

Designation of the substance	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
SULPHURETTED HYDROGEN, or **HYDRIC SULPHIDE,** H_2S: A colorless gas, having the fetid odor of rotten eggs.	Blast furnace plants, in granulating the slag; distillation of sulphur waters; ultramarine works; Leblanc soda and chemical factories; in the manufacture of the compounds of sulphur and phosphorus; sulphur metals (manufacture and use); sulphide of soda and sulphide of barium industry (manufacture of sulphide colors and dyeing with these); the extraction of cellulose (straw and wood); in the waste waters of industries which make use of organic substances; sedimentation tanks of sugar works; precipitation of soda residua containing calcium sulphide; work in sewers, latrines, and dung pits; illuminating-gas plants; flax retteries; tanneries.	In the form of gas, through the respiratory organs, as pure hydric sulphide gas; often found in admixture with other gases (with CO_2, N, NH_4, and carburetted hydrogen); direct action on the conjunctiva.	**Acute Poisoning.**—In the less violent cases there are gastric distress, nausea, fetid eructations, irritation and inflammation of the conjunctiva; rarely, erosion of the cornea, formation of vesicles on the lips, irritating cough, headache, and a sensation of giddiness. In long-continued inhalation convulsions and paralyses occur. In severe cases there are contraction of the pupils, slowing of the pulse, Cheyne-Stokes respiration, nystagmus, trismus, and tetanus. With a very high proportion of sulphuretted hydrogen in the air a man suddenly falls, becomes unconscious and dies without convulsions (apoplectic form). **Chronic Poisoning.**—Conjunctival catarrh; a sense of pressure in the head and on the chest; headache, debility, vertigo, nausea, disturbances of digestion; sallow complexion and emaciation; slowing of the pulse; tendency to the formation of boils.

Special measures of relief: Before emptying of dung pits and the like, their contents should be thoroughly mixed with iron sulphate (5 kg. pro 1 cbm.); the emptying should be effected by mechanical apparatus; safety ropes to be attached to the workmen; prompt hoisting out of the unconscious workmen; removal of the soiled clothing; artificial respiration; administration of oxygen; hypodermics of ether or camphor.

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LIST OF INDUSTRIAL POISONS—*Concluded*

Designation of the substances	Branches of industry in which poisoning occurs	Mode of entrance into the body	Symptoms of poisoning
SULPHURIC ACID, H_2SO_4: A colorless, odorless, thick, oily, fluid.	Manufacture of sulphuric acid; accumulator factories (mould and charging rooms); burnishing of iron, steel, etc.; textile industry, hat factories; petroleum distillation; factories for the manufacture of powdered fertilizers.	In the form of vapor, through the respiratory organs.	Inflammatory disease of the respiratory organs (acute and chronic catarrh), inflammation of the lungs; anorexia; decalcification of the bones (according to Lewin); injury to the teeth through softening of the dentine. As a result of the bespattering of the skin with concentrated H_2SO_4 there is severe pain, a whitish discoloration of the skin, becoming brownish, with reddening and swelling of the surrounding tissues; in cases of extensive scalds there are, ultimately decomposition of the blood, formation of ulcers. of the duodenum, somnolence, and even death.
TAR: A product obtained by dry distillation, particularly of anthracite coal and lignite.	Manufacture of illuminating gas; coke ovens; tar works; tar-product factories; plants for wood preserving; manufacture of roofing paper; use for concrete paving; painting of metals; as a fuel; briquet factories.	It acts on the skin; in the form of vapor, on the respiratory organs.	Tar itch under the form of diffuse acne, eczema or psoriasis, primarily on the upper extremities, later, also, on the other parts of the body; not infrequently on the irritated portions of the skin there appear cancroid ulcers, especially of the scrotum (among chimney sweepers, paraffine and soot workers and briquet makers). Together with the effect on the greater portion of the skin, there are also general symptoms: Loss of appetite, nausea, diarrhea, headache, numbness, vertigo, besides disturbances of the urinary bladder (ischuria, strangury), also albuminuria and œdema.
TURPENTINE OIL: A mixture of various terebinthine hydrocarbons, $C_{10}H_{16}$, differing in odor and in composition according to the botanical species from which they are severally derived.	Manufacture of varnish, cement, lacquer, sealing wax, colors; tapestry printing; trade of decorator, lacquerer and house painter; as a cleansing agent in various industries.	In the form of vapor, it acts upon the mucous membranes; in fluid state, it acts on the epidermis.	Irritation of the mucous membrane of the eyes, of the nose (coryza), and of the upper air passages (hemming, cough, bronchial inflammation); salivation; besides there are insensitiveness, giddiness, headache. Prolonged action of the oil causes irritation of the kidneys, and then these organs excrete urine having the odor of violets. Severe irritation of the skin is excited, especially by the so-called pine oil (Russian oil of turpentine).

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List of Industries in Which Poisoning May Occur*

Branches of industry in which poisoning may occur	Designation of industrial poison
Abelite, manufacture of	Nitrous gases.
Accumulator, electrical, works	See Batteries, storage.
Acetate of lead, manufacture of	Lead.
Acetylene production (if calcium carbide contains admixture of calcium phosphate)	Phosphureted hydrogen.
Acids, commercial manufacture of	Arsenic.
Acid, hydrochloric, manufacture of	Hydrochloric acid.
Acid, hydrofluoric, manufacture of	Hydrofluoric acid.
Acid, muriatic, manufacture of	Hydrochloric acid.
Acid, picric, manufacture of	Picric acid.
Acid, stearic, manufacture of	Acrolein.
Acid, sulphuric, manufacture of	Nitrous gases, sulphur dioxide.
Acid, valeric, manufacture of	Amyl alcohol.
Air pollution	Carbon dioxide, carbon monoxide.
Alcohol, denatured	Benzol, methyl alcohol, pyridine.
Alkaloids, manufacture of	Benzol.
Amber workers	Lead.
Amalgam	Mercury.
Ammonia salts, manufacture of	Ammonia.
Ammunition, manufacture of	Acrolein, antimony, lead (see also "Explosives").
Amid compounds of benzol, etc.	Aniline.
Amyl nitrite, manufacture of	Amyl alcohol.
Anatomical preparations	Formaldehyde, phenol.
Aniline color dye factories: Aniline orange, aurantia, saffron yellow, Manchester yellow, Meldola dyes, corvulin, Bismarck blue, indulin, fast black	Aniline, arseniureted hydrogen, antimony, hydrochloric acid, methyl bromide, nitrobenzol, nitrous gases.
Antimony alloys, and extraction of	Antimony, lead.
Antiseptic dressing, manufacture of	Mercury, phenol.
Antipyrine, manufacture of	Benzine, phenylhydrazine.
Arsenic acid, manufacture of	Arsenic, arseniureted hydrogen.
Arsenic mining	Arsenic
Arsenical ores, smelting	Arsenic
Artificial flowers and leaves	Arsenic, lead.
Artificial ice and cold storage	Ammonia.
Asphalt, testing	Carbon disulphide.
Aurantia dyes	Aniline dyestuffs.
Automobilists	Carbon monoxide, benzine.
Babbiting metal, and solder	Lead.
Bakers and confectioners	Carbon dioxide, carbon monoxide.
Balloon filling with impure hydrogen gas	Arseniureted hydrogen.
Barium sulphide, manufacture of	Sulphureted hydrogen.
Barometers, manufacture of	Mercury.
Batteries, storage, dry, manufacture of	Benzol, creosote, hydrochloric acid, sulphuric acid, lead, mercury, pitch, zinc chloride.
Batteries, storage, wet, manufacture of	Chromium compounds.
Beet sugar, manufacture of	Ammonia, sulphureted hydrogen.
Benzine plants	Benzine.
Benzol	Benzol.
Bicycles, manufacture of	Amyl acetate.
Bismarck blue, manufacture of	Aniline dyestuffs.
Bismuth, manufacture of	Arsenic.
Black aniline colors	Aniline dyestuffs.
Blacksmiths	Acrolein, carbon monoxide, cyanogen compounds.
Blast furnace workers	Carbon monoxide, cyanogen compounds, lead fumes, if lead is present in iron ore, sulphur dioxide, sulphureted hydrogen (in granulating slag).
Bleacheries	Chlorine, sulphur dioxide.
Bleaching agents, manufacture of	Nitrous gases.
Bleaching agents, for bristles, cane, silk, straw hats, wax and wool	Sulphur dioxide.
Bleaching agents for cotton, linen and paper	Chloride of lime.
Bleaching agents for cane and extraction of its silicates	Hydrofluoric acid.
Bleaching agents for fats, oil and wax	Chlorine, chromium compounds.
Bone ash, refuse of meat extract	Phosphorus.
Bone-black, refuse of sugar refineries	Phosphorus.
Bone-black, manufacture of	Ammonia, phosphorus.
Bone, extraction of	Sulphur dioxide.
Bone, rendering plants	Acrolein, benzine.
Bone tar, manufacture of	Pyridin.
Bookbinders	Carbon monoxide, methyl alcohol.
Boot and shoe industry	Benzine, methyl alcohol, lead, mercury.
Bottle caps and capsules	Lead.
Box and card factories	Arsenic, chrome and lead compounds.
Brasiers	Carbon monoxide.
Brass etching	Arsenic chloride.

*Arranged by Geo. M. Kober, M. D.

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LIST OF INDUSTRIES AND POISONS.—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Brass instruments, musical	Lead.
Brass foundries	Antimony, benzine, carbon dioxide, carbon monoxide, lead, phosphorus, sulphur dioxide, zinc fumes.
Brass polishing	Lead, oxalic acid, sulphuric acid.
Brass lacquer	Amyl acetate, methyl alcohol.
Breweries, fermentation rooms	Carbon dioxide.
Breweries, fumigation of vats, and disinfection	Sulphur dioxide, zinc oxide.
Breweries, shellacing casks	Methyl alcohol.
Brick kilns, brick and tile makers	Carbon dioxide, carbon monoxide, sulphur dioxide, lead glaze.
Briquet factories for fuel	Tar.
Britannia metal	Antimony.
Bronze workers	Antimony, lead, zinc, arsenic, acids, phosphorus.
Bronzing with nitrate of mercury	Mercury.
Brown mineral mills	Manganese.
Brunswick green	Arsenic.
Brush makers	Anthrax, lead, methyl alcohol, tar (see also Bleaching).
Bullets, dipping	Acrolein.
Bullets, manufacture of	Antimony, lead.
Burnishing of iron and steel	Antimony, sulphuric acid.
Cable wire, manufacture of	Carbon disulphide, lead.
Cabinet makers	Aniline stains, chrome lead stains.
Caoutchouc solvent and refining of	Benzol, carbon disulphide, sulphur chloride.
Caisson work	Carbon dioxide.
Calcining dolemite, etc.	Carbon dioxide, carbon monoxide.
Calico printing	Aniline, chromium, cyanogen and chlorine compounds, hydrochloric acid, lead, methyl alcohol, phenol, antimony, arsenic, carbon monoxide.
Candles, manufacture of	Sulphur dioxide.
Cane factories	Aniline stains, chlorine, chloride of lime, hydrofluoric acid, methyl alcohol, sulphur dioxide.
Canning industry	Carbon monoxide, lead, acid fumes, sulphur dioxide.
Carbolic acid	Phenol.
Carbonated waters	Carbon dioxide.
Carbon chloride, manufacture of	Carbon disulphide.
Carbon sulphurate, manufacture of	Carbon disulphide.
Carbonizing of materials	Acid fumes and arseniureted hydrogen.
Carpet cleaning	Benzine.
Carpet dye	Arsenic.
Cassel green	Arsenic.
Celluloid manufacture	Acetaldehyde, aniline and lead colors, cyanogen compounds, methyl alcohol, nitrous gases, sulphureted hydrogen.
Cellulose, extraction from straw and wood	Nitrous gases, sulphureted hydrogen.
Cements	Turpentine, benzine.
Ceramic industry	Hydrofluoric acid, lead, sulphur dioxide (see also Potteries).
Cerium, preparation of	Nitrous gases.
Chair factories, polishing	Methyl alcohol, petroleum (see also Rattan Industry).
Chalk, colored	Arsenic.
Charcoal burning	Carbon monoxide.
Chemical cleansing establishments	Benzine, benzol.
Chemical cleansing removal of ink and rust stains	Oxalic acid.
Chemical industry	Ammonia, aniline, carbon disulphide, chlorine, cyanogen compounds, hydrochloric acid, methyl compounds, nitrous gases, nitrobenzol, phosphorus sesquisulphide, picric acid, sulphur dioxide, carbon monoxide, etc.
Chloride of lead	Lead.
Chloride of lime, manufacture of	Chlorine, arseniureted hydrogen.
Chlorinating process	Chlorine.
Chlorine, organic products	Chlorine.
Chloroform manufacture	Chloride of lime.
Chromate of lead	Chomium, lead.
Chromate tanning	Chromium compounds.
Chromium colors and preparations	Chromium compounds.
Chromo-lithography	Arsenic, brass, chromium, lead, nitrous gases in etching, turpentine.
Christmas ornaments, manufacture of	Arsenic.
Chrysoidin fast black, manufacture	Aniline dyestuffs.
Church crosses, gilding	Mercury.
Cinnabar	Mercury.
Cleaning, dry	Benzine, benzol.
Coal mines	Carbon dioxide, carbon monoxide (see mining).
Coal oil	Petroleum.
Coal-tar anthracite distillation	Phenol, pyridine, tar.

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LIST OF INDUSTRIES AND POISONS—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Coal-tar color industry	Aniline, formaldehyde, methyl alcohol, nitrobenzol, nitrous gases.
Cochineal	Arsenic.
Coke ovens	Ammonia, carbon monoxide, tar.
Collodion cotton	Nitrous gases.
Commercial acids, impure	Arsenic.
Colors, manufacture of for paints, etc.	Benzine, benzol, chromium compounds, arsenic, lead, mercury, turpentine.
Colored chalk	Arsenic.
Colored lights	Arsenic, antimony.
Colored paper	Arsenic, chromium, lead compounds.
Colored pencils	Aniline dyestuffs.
Combs, horn-celluloid	Acetaldehyde, acid fumes, aniline, lead colors (see also Celluloid).
Compositors	Lead, antimony, arsenic, benzine.
Concrete paving	Tar.
Coopers	Methyl alcohol shellac.
Copper plate etching and engraving	Nitrous gases.
Copper polishing	Oxalic acid.
Copper smelting	Arsenic, carbon monoxide, sulphur dioxide.
Copper workers	Arseniureted hydrogen, lead, nitric and sulphuric acid fumes.
Corvuline dye	Aniline dyestuffs.
Cowper apparatus	Carbon monoxide.
Creasote, cresol	Phenol.
Cumol	Benzol.
Cutlery industry	Carbon monoxide, acid fumes, lead (see also Brass, Tempering, Tinning).
Decorators and painters	Arsenic, benzine, chromium compounds, lead, mercury, methyl alcohol, turpentine.
Decomposition gases	Ammonia, carbon dioxide, sulphureted hydrogen.
Denaturing of spirits	Methyl alcohol, pyridine.
Dentists	Mercury.
Deoxidating processes	Nitrous gases.
Diamond cutting and setting of precious stones	Lead, carbon monoxide.
Dinitrobenzol, manufacture of	Nitrobenzol.
Dinitrochlorobenzol, manufacture of	Nitrobenzol.
Dinitro-compounds, manufacture of	Nitrobenzol.
Dip for scabby sheep	Arsenic.
Disinfection	Carbon disulphide, chlorine, chloride of lime, cyanogen compounds, formaldehyde, mercury bichloride, phenol, sulphur dioxide.
Distilleries	Carbon dioxide, sulphureted hydrogen, sulphur dioxide.
Dolemite calcining	Carbon dioxide, carbon monoxide.
Drying processes by means of open fires	Carbon monoxide.
Dung pits	Ammonia, sulphureted hydrogen.
Dyes, antiseptic	Mercury.
Dyes, organic, manufacture of	Acridine.
Dyestuffs	Ammonia, chloride of lime (see also Aniline Dyestuffs).
Dyeing and printing, fixer and mordant	Nitrous gases.
Dyeing and dye works	Antimony, arsenic, aniline dyestuffs, benzol, chromium compounds, cyanogen compounds, hydrofluoric acid, phenol, oxalic acid, picric acid, sulphuretted hydrogen (dyeing with sulphide colors), ammonia, lead, methyl alcohol.
Dynamite, manufacture of	Nitrous gases, nitroglycerine.
Electrical accumulator works	See Batteries.
Electric lamps, manufacture of	Lead, mercury.
Electric lamps, incandescent wire	Amyl acetate.
Electric line workers.	Carbon monoxide, solder.
Electric meters.	Mercury, lead (see also Brass Industry).
Electroplating.	Cyanogen compounds.
Electrotyping	Antimony, arsenic, lead, carbon monoxide.
Emery wheels, babbitting of	Lead.
Enamelling works	Hydrochloric acid, lead, benzine, carbon monoxide.
Engraving, steel	Mercury.
Essences, fruit, artificial	Amyl alcohol.
Etching on brass	Arsenic chloride, nitrous gases.
Etching on metals	Arseniureted hydrogen, mercury, nitrous fumes, chlorine, phosphoric acid.
Ether, methyl	Dimethyl sulphate.
Ethyl violet	Aniline dyestuffs.
Extraction of antimony	Antimony.
Extraction of bone	Sulphur dioxide.
Extraction of gold and silver	Cyanogen compounds, mercury.
Explosives, manufacture of	Aniline dyestuffs, mercury, nitrous gases, nitrobenzol, nitroglycerine, picric acid.

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LIST OF INDUSTRIES AND POISONS—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Farmers	Carbon dioxide in Silos. See also insecticides
Fats, bleaching of	Chromium compounds.
Fats, extraction of	Benzine, benzol, acrolein, carbon disulphide.
Fats, solvents	Benzol, benzine, carbon disulphide, sulphur chloride.
Faucets, brass, polishing	Lead.
Feathers, ornamental	Benzine.
Fermentation rooms	Carbon dioxide.
Felt-hat industry	Mercury, methyl alcohol, nitrous gases, sulphuric acid, nitric acid, arsenic dyestuffs, carbon monoxide.
Ferrosilicon	Arseniureted and phosphureted hydrogen.
Fertilizers, artificial manufacture of	Hydrochloric acid, hydrofluoric acid, sulphuric acid, sulphureted hydrogen, benzine.
File cutting	Lead.
Fireworks	Antimony, arsenic, carbon monoxide, phosphorus.
Firearms, manufacture of	Antimony, carbon monoxide, nitrous gases.
Firemen	Benzine, carbon monoxide, nitrous and other acid fumes.
Flasks, manufacture of	Lead.
Flax retteries	Sulphureted hydrogen.
Flowers, artificial	Arsenic, lead.
Foundries, iron	Carbon monoxide, sulphuric acid.
Fluoric acid	Hydrofluoric acid.
Fluorides, extraction of	Hydrofluoric acid.
Fruit essences, manufacture of	Amyl alcohol.
Fruit, dried, preservation	Sulphur dioxide.
Fuel briquet factories	Tar.
Fumigation casks, hops, fruit	Sulphur dioxide.
Furnace gases	Carbon monoxide, sulphur dioxide (see also Blast Furnaces).
Furniture factories, staining and polishing	Aniline, arsenic, chrome stains, lead, methyl alcohol, petroleum, phenol, turpentine.
Furriers	Lead for dyeing; mercury and nitrous gases fo rabbit fur, arsenic, anthrax.
Galvano-plasty	Cyanogen compounds.
Galvano-techniques	Nitrous gases.
Galvanizing with zinc or tin	Ammonia, arseniureted hydrogen, hydrochloric and sulphuric acids, zinc.
Garage workers	Benzine, carbon monoxide.
Garbage fat extraction	Benzine.
Gardeners	See Insecticides.
Garment workers	Aniline and arsenic dyes, carbon monoxide from ironing stoves, lead from weighted silk.
Gas plants	Ammonia, carbon monoxide, cyanogen compounds, tar.
Gas and steam fitters	Arseniureted hydrogen, lead, nitrous gases.
Gas machines	Carbon monoxide.
Gas purification	Carbon monoxide, cyanogen compounds.
Gasoline	Benzine.
Gelatine manufacture	Sulphur dioxide.
Gilding and silvering	Mercury.
Glass etching	Hydrofluoric acid.
Glass factories	Arsenic, hydrofluoric acid, hydrochloric acid chromium compounds, carbon monoxide, lead, manganese, phenol (see also Painters).
Glass polishing	Lead.
Glaze mixing and dipping	Lead.
Glove and mitten manufacture	Anthrax,acids,aniline, chrome and lead compounds.
Glove cleaning	Benzine.
Glucose, manufacture of	Arsenic from impure sulphuric acid.
Glue, manufacture of	Sulphur dioxide, chloride of lime.
Glycerine, trinitrate	Nitroglycerine.
Gold, extraction of	Cyanogen compounds, mercury.
Gold plating	Cyanogen compounds.
Grease removal	Benzine, benzene, carbon disulphide.
Gums, solvent for	Carbon disulphide.
Gun cotton	Nitroglycerine, nitrous gases.
Gunsmiths	Antimony, cyanogen compounds, carbon monoxide.
Hair industry	Anthrax.
Hardening and tempering steel magnets, piano wire, springs, files, etc.	Acrolein, cyanogen compounds and lead.
Hat, felt, factory	Mercury, methyl alcohol, nitrous gases, sulphuric acid, arsenic, dyestuffs, carbon monoxide.
Hat, straw, factory	Sulphur dioxide, methyl alcohol, oxalic acid.
Heating and power plants	Carbon dioxide, carbon monoxide.
Hectograph composition	Chromium compounds, aniline.
Hides and skins	Anthrax, arsenic, sulphur dioxide(see alsoTanning).
Hittorf tubes	Mercury.

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LIST OF INDUSTRIES AND POISONS—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Hydrochloric acid	Nitrous gases.
Hydrogen gas	Arseniureted hydrogen.
House painting	Arsenic, benzine, lead, chrome colors, methyl alcohol, turpentine.
Ice machines	Ammonia, sulphur dioxide.
Igniting agents	Phosphorus.
Illuminating gas, manufacture of	Ammonia, benzol, carbon monoxide, carbon disulphide, sulphureted hydrogen, tar.
Imitation bitter-almond oil	Nitrobenzol.
Imitation silk factories	Carbon disulphide, ammonium sulphide, nitrous fumes.
Imperial yellow dye, manufacture of	Aniline dyestuffs.
Impregnated wood	Phenol, tar.
Incandescent electric light	Amyl acetate, carbon monoxide, mercury, methyl alcohol.
India-rubber industry	Aniline oil, antimony, benzine, benzol, carbon disulphide,, cinnabar (mercury), hydrochloric acid, lead, sulphur dioxide and chloride, tar, wood alcohol.
Indian white fire	Arsenic.
Indulin dye, manufacture of	Aniline dyestuffs.
Ink stains, removal of	Oxalic acid.
Insecticides, manufacture and use of	Arsenic, carbon disulphide, cyanogen and mercury compounds, sulphur dioxide.
Insulated wire, manufacture of	Carbon disulphide, lead.
Iodine, manufacture of	Benzol.
Iron chloride, sulphate, manufacture	Nitrous gases.
Iron, deoxidation of	Nitrous gases.
Iron, galvanizing with zinc or tin	Ammonia, arseniureted hydrogen, acid fumes and zinc.
Iron silicate, impure, decomposition of	Arseniureted and phosphureted hydrogen.
Iron sulphate, manufacture of	Arseniureted hydrogen.
Ironing	Carbon monoxide, chlorine, arsenic
Iron sanitary ware	Carbon monoxide, lead, acid fumes.
Iron and steel workers	Carbon monoxide, other furnace gases (see also Cutlery Industry).
Jewelry, manufacture of	Ammonia, amyl acetate, cyanogen compounds, lead solder, hydrochloric, nitric and sulphuric acids, mercury, carbon monoxide (see also Brass).
Kaiser green	Arsenic.
Krems white	Lead.
Lace workers	Carbon monoxide.
Lacquer manufacture	Ammonia, amyl acetate, benzine, benzol, methyl alcohol, turpentine.
Lampblack, manufacture	Phenol.
Lamp shades, coloring purposes	Arsenic.
Lanolin, extraction of	Carbon disulphide.
Lard making	Acrolein, ammonium sulphide, acid fumes.
Latrines	Ammonia, sulphureted hydrogen.
Laundries	Benzine, benzol, chlorine, aniline colors for marking ink, carbon monoxide, arsenic from coke-burning ironing stoves.
Lead alloys	Antimony, copper, tin, etc.
Lead colors	Lead.
Lead, deoxidation of	Nitrous gases.
Lead metal	Arsenic.
Lead smelting	Antimony, arsenic, lead, sulphur dioxide.
Lead plating	Arseniureted hydrogen.
Leaf-metal workers	Ammonia, amyl acetate, acetone, benzine, benzol, methyl alcohol, turpentine.
Leather industry	Arsenic, chromium compounds, lead, mineral acids
Leather sole stitching	Mercury.
Leather patent	Amyl acetate, benzine, methyl alcohol.
Leblanc soda, manufacture	Sulphureted hydrogen.
Ligroine	Benzine.
Lime chloride, manufacture of	Chlorine, arseniureted hydrogen.
Lime kilns	Carbon dioxide, carbon monoxide, sulphur-dioxide.
Linoleum, manufacture of	Acrolein, amyl acetate, arsenical, mercurial and lead pigments, benzine and turpentine, manganese, zinc oxide.
Linotyping	Antimony, arsenic, lead, organic vapors.
Litharge	Lead.
Lithographing	Arsenic, acid fumes, bronze powder, aniline, benzine, turpentine.
Litho-transfer work	Lead.
Lyddite, manufacture of	Picric acid.
Lysol	Phenol.

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LIST OF INDUSTRIES AND POISONS—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Manchester yellow, manufacture of	Aniline dyestuffs.
Manganese mills	Manganese.
Manumeters, manufacture of	Mercury.
Marble polishers	Lead.
Masonic white leather aprons	Lead.
Mattress, manufacture of	Anthrax, infectious diseases.
Matches, manufacture of	Chromium compounds, phosphorus.
Meldola dyes	Aniline dyestuffs.
Melinite, manufacture of	Picric acid.
Mercury compounds, manufacture of	Mercury.
Mercury mining	Mercury.
Mercury smelting	Mercury.
Mercury vapor lamps	Mercury.
Metal dipping	Acid fumes.
Metal burnishing	Antimony, acid fumes.
Metal etching	Arseniureted hydrogen, nitrous fumes, mercury.
Metal lacquer	Amyl acetate.
Metal polishing	Oxalic acid.
Metal refining	Nitrous gases.
Meters, electric, manufacture of	Mercury, lead (see also Brass Industry).
Methyl amines	Dimethyl sulphate.
Methyl esters	Dimethyl sulphate.
Methyl ether	Dimethyl sulphate.
Methyl violet	Aniline dyestuffs.
Methylizing of every kind	Diazomethane.
Mining	Arsenic, carbon dioxide, carbon monoxide, lead, mercury, nitroglycerine, nitrous fumes, sulphureted hydrogen, and other gaseous products of combustion of explosive compounds.
Mineral water, carbonated	Carbon dioxide.
Mints	Nitrous gases.
Mirbane oil	Nitrobenzol.
Mirror plating	Mercury.
Mirror silvering	Acetaldehyde, ammonia; lead, if backed with red lead.
Moulds, drying	Carbon monoxide.
Monotyping	Antimony, arsenic, acrolein, lead.
Moulding, picture-frame manufacture	Amyl acetate, bronze, methyl alcohol (see also Leaf-metal Workers).
Mordant in dyeing	Antimony, chromium compounds, etc.
Mosaic works	Manganese.
Muriatic acid	Hydrochloric acid.
Muslin green, color	Arsenic.
Naphtha, naphthol nitrates	Benzine, benzol, nitrous gases.
Naphthalein	Aniline, aniline dyestuffs.
Navy	Carbon monoxide, gun firing, and furnace rooms.
Nickel buffers and polishers	Lead, nickel-carbonyl.
Nickel platers	Benzene, lime, nickel salts, petroleum.
Neuwied green	Arsenic.
Nitric acid manufacture, salts and storage	Nitrous gases.
Nitrite of amyl	Amyl alcohol.
Nitrificating in chemical works	Nitrous gases.
Nitrobenzol	Aniline, nitrous gases.
Nitrocellulose	Nitroglycerine, nitrous gases.
Nitroglycerine	Nitrous gases.
Nitrojute	Nitrous gases.
Nitromannite	Nitrous gases.
Nitronaphthalene	Nitrobenzol.
Nitrophenol	Nitrobenzol.
Nitrosaccharose	Nitrous gases.
Nitroso dyes	Aniline dyestuffs.
Nitrotoluol	Nitrobenzol.
Oil, bleaching of	Chromium compounds, nitrous gases.
Oil, solvent	Benzine, carbon disulphide.
Oilcloth, manufacture of	Acrolein, amyl acetate, arsenical and lead pigments.
Oil, vitreol	Sulphuric acid.
Open-fire heating	Carbon monoxide.
Organ builders	Lead, bronze, methyl alcohol.
Organic dyes, manufacture	Acridine, arsenic.
Organic preparations, manufacture	Formaldehyde, phenylhydrazine, phosgene.
Oxalic acid, manufacture of	Oxalic acid.
Oxygen, manufacture of	Chloride of lime.
Painters and commercial artists	Arsenic, benzine, benzol, lead, mercury, methyl alcohol, tar, turpentine, phenol, amyl acetate, carbon disulphide.
Paper deoxidation	Nitrous gases.
Paperhangers	Arsenic, lead.

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LIST OF INDUSTRIES AND POISONS—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Paper mills	Chlorine, lead, sulphur dioxide, toxic color pigments.
Paraffine refining	Carbon disulphide.
Paris green	Arsenic.
Parrot green	Arsenic.
Paving material	Asphalt, tar.
Pencils, colored	Aniline dyestuffs.
Percussion caps	Mercury fulminate.
Perfumes, manufacture of	Dimethyl sulphate, methyl alcohol, nitrobenzol.
Petroleum industry, distillation and refining	Petroleum, sulphuric acid, hydrochloric acid chloride of lime, sulphur dioxide, lead, tar.
Pharmaceutical preparations	Mercury, methyl alcohol, nitrobenzol, etc.
Phenol nitrates, manufacture of	Aniline dyestuffs, nitrous gases, phenol.
Phenylhydrazine, manufacture of and its use for production of organic compounds	Phenylhydrazine.
Phosgene, manufacture of and its use for production of organic compounds	Phosgene.
Phosphor bronze	Phosphorus.
Phosphorus extraction from phosphorites and coprolites	Phosphorus and hydrofluoric acid.
Phosphorus, manufacture of	Benzol, phosphorus.
Phosphorus, red, manufacture of	Phosphureted hydrogen.
Phosphorus, sesquisulphide, manufacture of	Phosphureted hydrogen.
Phosphorus and sulphur compounds	Sulphureted hydrogen.
Photoengravers	Ammonium dichromate, nitrous fumes.
Photogen factories	Phenol.
Photographing establishments, material	Aniline colors, bromine compounds, cyanogen compounds, mercury, metol, chromium compounds, lead in retouching high lights.
Physical apparatus, manufacture of	Mercury, arseniureted hydrogen.
Pianos, manufacture of	Bronze, lead, methyl alcohol.
Picric acid manufacture	Aniline dyestuffs, nitrous gases, picric acid, phenol.
Picture frames, manufacture of	Bronze, amyl acetate, methyl alcohol (see Leaf-metal Workers).
Plumbers	Arseniureted hydrogen, lead, carbon monoxide.
Polish for furniture	Petroleum, methyl alcohol.
Polish for metals	Oxalic acid.
Porcelain enamelled ware	Lead.
Potteries	Hydrofluoric acid, hydrochloric acid, lead, manganese, arsenic, chrome, carbon monoxide (see also painters.)
Printing establishments	Acrolein, antimony, benzine, lead, carbon monoxide, arsenic, methyl alcohol.
Preservative fluid for animal tissues	Formaldehyde, methyl alcohol.
Preservative for wood	Arsenical color pigments, phenol, tar.
Projectiles, manufacture of, filling shops	Picric acid (see also Explosives).
Putty making	Lead.
Putrefaction processes, gases of	Ammonia, carbon dioxide, sulphureted hydrogen.
Pyridin, manufacture of	Pyridin.
Pyrites	Arsenic, nitrous gases.
Pyrotechniques	Antimony, arsenic, phosphorus
Quicksilver	Mercury.
Rabbit fur for felt hats	Mercury, nitrous gases.
Rag and shoddy industry	Acid fumes, infectious diseases.
Rattan industry	Aniline stains, chlorine, chromium, hydrofluoric acid, methyl alcohol, sulphur dioxide.
Red lead	Lead.
Refrigeration plants	Ammonia.
Rendering plants	Acrolein, benzine, carbon disulphide.
Resin, distillation of	Carbon monoxide.
Resin, solvent for	Benzine.
Rifle barrel, burnishing	Antimony.
Röntgen tubes, manufacture of	Mercury.
Roofers	Lead, solder, tar.
Roofing paper, manufacture of	Tar.
Roof tiling manufacture	Lead, carbon monoxide and other furnace gases.
Rubber industry, including rubber toys	Aniline, antimony, arsenic, benzine, benzol, carbon disulphide, and tetrachloride, lead, phenol, sulphur dioxide, and chloride, tar, mercuric sulphide, methyl alcohol, turpentine
Rubber tires, assembling of	Carbon disulphide.
Rugs, manufacture, dyeing	Arsenic and other toxic dyestuffs.
Rust stains, removal of	Oxalic acid.
Saffron yellow dye	Aniline dyestuffs.
Salamanders, drying houses and plaster	Carbon monoxide.
Sal ammoniac	Ammonia.
Salts of mercury	Mercury.
Sanitary ware factories	Lead.

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LIST OF INDUSTRIES AND POISONS—*Continued*

Branches of industry in which poisoning may occur	Designation of industrial poison
Schweinfurth green	Arsenic.
Sealing wax, manufacture of	Turpentine.
Sewer cleaning	Ammonia, carbon dioxide, sulphureted hydrogen.
Sedimentation tanks	Carbon dioxide, sulphureted hydrogen.
Sewing machine manufacture	Amyl acetate.
Sheep dip manufacture	Arsenic.
Sheele's green	Arsenic.
Shellac, solvent for	Methyl alcohol.
Shoddy manufacture	Hydrochloric acid, sulphuric acid.
Shot manufacture	Antimony, arsenic, lead.
Shoe manufacture	Bensine, methyl alcohol.
Silk bleaching	Sulphur dioxide.
Silk imitation factories	Carbon disulphide, ammonium sulphide, nitrous fumes.
Silk weighting	Lead.
Silver extraction	Mercury, cyanogen compounds.
Silver metal	Arsenic, lead, antimony.
Silver plating	Cyanogen compounds, mercury.
Smelting furnaces	Carbon monoxide and other furnace gases.
Smelting lead	Lead.
Smelting mercury	Mercury.
Smelting-sulphur bearing ores	Sulphur dioxide.
Soap factories	Acrolein, nitrobenzol, sulphuric acid, pyridin, ammonia cyanide, sulphur, tar.
Soda carbonate, manufacture of	Ammonia.
Soda chloride, manufacture of	Hydrochloric acid, chlorine.
Soda sediment, manufacture of	Nitrous gases.
Soda sulphate, manufacture of	Arseniureted hydrogen, hydrochloric acid.
Soda sulphide, manufacture of	Sulphureted hydrogen.
Soda works	Sulphureted hydrogen, hydrochloric and sulphuric acids.
Soldering	Arseniureted hydrogen, carbon monoxide, hydrochloric acid, lead, nitrous fumes.
Staining wood	Aniline, chromium, methyl alcohol, phenol.
Stannic acetate	Hydrochloric acid.
Starch, manufacture of	(See Putrefaction Gases.)
Stamping designs on embroidery	Lead and rosin.
Stamping mills	Mercury, nitrous gases.
Stearic acid factories	Acrolein.
Stearin refining	Carbon disulphide.
Steel engraving	Mercury.
Steel burnishing	Antimony, sulphuric acid.
Stereotyping	Antimony, lead, carbon monoxide.
Storage batteries	See Batteries.
Stone and marble polishers	Lead.
Straw hats, bleaching	Sulphur dioxide.
Straw deoxidation	Nitrous gases.
Sugar, beet sugar	Ammonia.
Sugar plants, saturation vessels	Carbon dioxide.
Sugar refineries	Phosphorus, sulphureted hydrogen.
Sulphur, refining of	Benzol.
Sulphur metals, manufacture and use of	Sulphureted hydrogen.
Sulphur extraction in gas purification	Carbon disulphide
Sulphur solvent for	Carbon disulphide, sulphur chloride.
Sulphur, water distillation of	Sulphureted hydrogen.
Sulphur and phosphorus compounds, manufacture of	Sulphureted hydrogen.
Sulphide colors, manufacture and use of	Sulphureted hydrogen.
Sulphuric acid, manufacture of	Nitrous gases, sulphur dioxide.
Sulphurous acid and salts, manufacture of	Nitrous gases, sulphur dioxide.
Surgical dressings	Mercury, phenol.
Swiss green	Arsenic.
Tailors	See Garment Workers.
Tallow rendering plants	Acrolein, sulphuric acid.
Tallow refining	Carbon disulphide, chlorine, acid fumes.
Tanneries, tanning and leather dressing	Ammonia, anthrax, arsenic, carbon dioxide (in tan pits), chromium compounds, lead (white leather), sulphur dioxide, sulphureted hydrogen, acids, benzine, amyl acetate.
Tapestry printing	Turpentine, toxic color pigments.
Tar color industry	Aniline, chromium compounds, phosphorus, etc.
Tar works	Tar.
Taxidermy	Arsenic, carbon disulphide.
Telephone wire, manufacture of	Lead.
Tempering and hardening, steel magnets, piano wire, springs, files, etc.	Acrolein, cyanogen compounds, lead.
Textile fabrics, deoxidation of	Nitrous gases.
Textile industry	Arsenical colors, lead, sulphuric acid.
Textile printing	Antimony, arsenic, chromium, lead compounds.
Thermometers	Mercury.

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List of Industries and Poisons—*Concluded*

Branches of industry in which poisoning may occur	Designation of industrial poison
Thorium, preparation of	Nitrous gases.
Tin foil	Lead.
Tin ware and tin shops and tinning	Ammonia, arseniureted hydrogen, chlorine, carbon monoxide, hydrochloric acid, lead, sulphuric acid.
Tissue hardening and preserving	Formaldehyde.
Toluol, manufacture of	Benzol.
Toys, coloring of	Arsenic.
Toy balloons, filling	Arseniureted hydrogen.
Transfer chromos	Lead.
Turkey red, mordant for	Chromium compounds.
Typefounders	Acrolein, antimony, arsenic, lead.
Typesetters	Benzine, lead.
Ultramarine works	Sulphur dioxide, sulphureted hydrogen.
Upholstery	Anthrax and infectious diseases.
Valeric acid, manufacture of	Amyl alcohol.
Varnish, manufacture and use of	Acrolein, ammonia, benzine, lead, methyl alcohol, turpentine.
Vinegar, manufacture of	Acetaldehyde.
Vienna green and red	Arsenic.
Viscosine, manufacture of	Nitrous gases.
Vulcanizing and red dyeing of rubber	Antimony, arsenic, carbon disulphide (see also Rubber).
Wall-paper, manufacture of	Arsenic, lead (see also paper mills).
Wall-paper, hangers and scrapers	Arseniureted hydrogen.
Waste waters of industrial plants making use of organic matter	Sulphureted hydrogen.
Watch factories	Benzine, cyanogen compounds, lead for dials, nitrous gases (see also Brass and Tempering).
Water gas, carburizing	Benzol.
Water gilding	Mercury.
Waterproof material	See Rubber.
Wax bleaching	Chromium compounds, sulphur dioxide.
Wax refining	Carbon disulphide.
Weather vane gilding	Mercury.
Well gas	Carbon dioxide.
Whip, factories	See Rattan Industry.
White lead	Lead.
White metal	Antimony.
Window shades, green	Arsenic.
Wine cellars	Carbon dioxide.
Wine preserving	Sulphur dioxide.
Wire galvanizing with zinc	Ammonia arseniureted hydrogen, hydrochloric acid, sulphuric acid, zinc.
Wire tempering	Lead, acerolein, cyanogen compounds.
Wool bleaching	Sulphur dioxide.
Woolen refuse, deoxidation of	Nitrous gases.
Wood alcohol	Methyl alcohol.
Wood deoxidation of	Nitrous gases.
Wood impregnating and preserving	Arsenical paints, phenol, tar.
Wood staining and polishing	Aniline colors, chromium compounds, lead, arsenic colors, methyl alcohol, alcohol denatured with pyridin, phenol, petroleum.
Workrooms, crowded	Carbon dioxide.
Xyloidine, manufacture of	Nitrous gases.
Xylol	Benzol.
Yeast, compressed, factories	Carbon dioxide.
Zapone lacquer	Amyl acetate.
Zinc chloride, manufacture of	Arseniureted hydrogen.
Zinc deoxidation of	Nitrous gases.
Zinc ore smelting	Antimony, arsenic, carbon monoxide, lead, manganese, sulphur dioxide.
Zinc plating	Ammonia, arseniureted hydrogen, hydrochloric, sulphuric acids and zinc.
Zinc sulphate, manufacture of	Arseniureted hydrogen.
Zoological preparations	Formaldehyde.

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MORTALITY AND MORBIDITY STATISTICS*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
All occupations reported..	210,507	2.2	14.8	5.5	7.3	11.9	8.0	8.5	2.6	10.5				
10 to 14 years.........	176	4.0	4.0	....	1.1	2.3	8.5	1.1	1.7	42.0				
15 to 19 years.........	5,356	9.0	23.0	0.5	0.5	3.8	7.0	1.5	2.2	26.6				
20 to 24 years.........	12,197	7.6	31.7	0.9	0.6	3.5	6.3	2.1	3.4	21.6				
25 to 34 years.........	28,635	5.0	31.0	1.3	1.2	4.8	7.4	4.0	3.7	18.4				
35 to 44 years.........	32,461	2.7	23.6	3.3	2.7	7.7	9.5	6.6	3.7	14.4				
45 to 54 years.........	35,711	1.5	14.4	6.8	5.9	11.3	9.0	9.7	3.5	10.2				
55 to 64 years.........	35,953	0.7	7.5	9.4	10.1	15.4	8.3	11.7	2.3	6.3				
65 to 74 years.........	33,462	0.3	3.6	8.4	13.4	18.9	7.6	12.0	1.2	3.9				
75 to 84 years.........	20,802	0.1	1.3	5.6	15.1	18.5	7.0	10.5	0.5	3.1				
85 years and over......	5,464	....	0.6	3.1	12.5	14.1	6.3	7.7	0.3	3.4				
Unknown.............	290	0.7	8.3	2.8	5.2	7.2	4.5	2.8	6.6	41.0				
All occupations reported, females..............	27,459	2.8	21.0	8.1	5.9	10.3	7.0	7.3	1.6	3.2				
10 to 14 years.........	71	12.7	26.8	....	1.4	5.6	7.0	2.8	1.4	5.6				
15 to 19 years.........	2,198	9.2	33.3	0.7	0.4	4.7	6.0	2.6	3.0	5.3				
20 to 24 years.........	3,653	5.7	39.8	0.7	0.6	4.8	5.3	3.0	2.9	3.9				
25 to 34 years.........	5,141	3.3	35.7	3.1	1.3	6.4	5.7	4.6	2.4	3.0				
35 to 44 years.........	4,500	2.0	21.3	10.1	4.0	10.5	6.4	7.7	1.1	2.6				
45 to 54 years.........	4,398	1.0	10.2	15.6	8.4	12.7	8.0	10.0	0.9	2.9				
55 to 64 years.........	4,174	0.6	5.6	14.7	12.3	15.4	9.0	11.5	0.8	2.5				
65 to 74 years.........	1,887	0.3	3.2	9.5	13.7	17.5	9.1	11.1	0.3	2.9				
75 to 84 years.........	1,078	0.1	1.8	7.0	15.1	16.3	9.0	9.2	0.4	4.4				
85 years and over......	338	....	....	4.1	11.5	12.4	5.0	7.1	....	4.1				
Unknown.............	21	4.8	4.8	4.8	19.0	9.5	....	....	4.8	4.8				
Agricultural pursuits, males:														
25 to 34 years.........	3,711	7.9	26.2	1.6	1.4	4.6	6.3	2.7	4.2	18.3				
35 to 44 years.........	4,138	4.5	19.3	4.5	2.6	7.4	8.4	4.7	5.4	15.4				
45 to 54 years.........	5,816	2.4	12.3	8.4	5.9	11.9	8.4	6.9	3.9	9.1				
55 to 64 years.........	8,424	1.1	6.7	10.1	11.5	16.8	7.6	9.0	2.3	6.1				
Agricultural laborers.....	....	....	....	....	....	....	....	....	....	....	1020	1159	1.34	0.72
25 to 34 years.........	1,800	7.1	28.6	1.0	1.6	4.8	6.5	2.1	5.2	20.2				
35 to 44 years.........	1,701	2.8	20.0	2.9	2.5	8.5	10.0	4.4	5.6	18.0				
45 to 54 years.........	1,958	1.8	14.9	5.1	5.3	12.8	10.6	5.7	3.2	9.6				
55 to 64 years.........	2,269	0.7	8.4	7.6	11.5	16.9	8.8	9.6	2.4	7.4				

* The mortality statistics showing the number and per cent. distribution, by important causes, of deaths of persons at least 10 years of age engaged in certain specified occupations, classified by age, for the registration area in the United States, are based upon data published in Bulletin 108. Bureau of the Census, in 1909.

[1] The morbidity statistics are based upon a study of sickness and mortality hazards in certain occupations, compiled from the experience of the Local Sick Benefit Society of Leipsic, 1887 and 1904, by Dr. Lee K. Frankel, Vice-President Metropolitan Life Insurance Company, New York, and presented before the Detroit Conference, Niagara Falls, September 4, 1913.

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Farmers, planters, and overseers:														
25 to 34 years	1628	9.2	24.9	2.3	1.4	4.6	6.4	3.3	3.1	13.4				
35 to 44 years	2091	6.0	18.8	5.5	2.8	6.6	7.4	5.0	5.2	11.7				
45 to 54 years	3366	2.9	11.0	10.4	6.2	11.5	7.0	7.4	4.2	8.2				
55 to 64 years	5589	1.2	6.1	11.0	11.5	17.1	7.1	8.8	2.3	5.4				
Gardeners, florists, nurserymen, etc.:														
25 to 34 years	69	5.8	23.2	1.4	1.4	4.3	8.7	4.3	4.3	13.0				
35 to 44 years	96	4.2	33.3	7.3	3.1	2.1	9.4	5.2	5.2	7.3				
45 to 54 years	184	1.1	15.2	6.5	5.5	14.7	11.4	9.2	3.8	5.4				
55 to 64 years	223	0.4	7.2	13.0	7.6	16.1	9.9	10.3	2.7	5.8				
Agents—Transportation:														
25 to 34 years	262	5.7	32.8	2.3	0.4	5.3	7.6	3.8	6.1	10.7				
35 to 44 years	366	4.4	18.9	5.2	1.4	7.7	7.4	7.1	7.4	7.6				
45 to 54 years	552	1.6	8.0	9.6	6.9	11.4	7.4	12.0	5.3	6.9				
55 to 64 years	650	0.8	4.9	10.2	12.0	15.7	6.6	10.9	4.5	3.7				
Aluminum and brass factory workers											761	....	0.50	
Artificial ice makers											701	....	0.56	
Asphalters											1317	....	0.88	
Asphalt and concrete workers											1320	....	0.71	
Bakers	952	....	....	....	....	....	....	....	....	....	497	....	0.15	
25 to 34 years	141	3.5	29.1	1.4	0.7	1.4	12.1	2.8	5.7	12.8				
35 to 44 years	176	1.1	29.0	4.0	1.7	10.2	7.4	8.5	4.5	5.7				
45 to 54 years	198	3.0	18.7	9.6	4.5	9.6	8.6	10.6	4.5	5.6				
55 to 64 years	181	0.6	9.4	6.6	8.8	17.7	8.3	10.5	5.0	3.3				
Bankers and brokers	712													
25 to 34 years	74	2.8	36.1	....	....	2.8	11.1	8.3	5.6	5.6				
35 to 44 years	119	6.8	12.2	4.1	2.7	2.7	14.9	5.4	8.1	5.4				
45 to 54 years	174	2.5	3.4	10.9	10.1	10.1	8.4	17.6	4.2	2.5				
55 to 64 years	165	....	4.6	7.5	9.8	14.9	9.8	14.4	3.4	3.4				
Barbers and hairdressers											395	....	0.32	
25 to 34 years	283	3.9	40.6	1.1	0.4	7.1	4.2	2.5	4.6	7.1				
35 to 44 years	345	2.6	25.8	2.6	2.6	8.4	6.1	9.6	4.6	6.9				
45 to 54 years	314	1.3	16.2	4.8	7.3	12.4	6.4	9.6	3.2	7.0				
55 to 64 years	180	0.6	11.7	6.7	8.9	19.4	3.3	12.8	1.7	3.3				

MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations: All causes	Per cent. of all causes at specified age: Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bight's disease	Suicide	Accident	No. of days of sickness per annum per 100 members[1]: Males	Females	No. of deaths per 100 members[1]: Males	Females
Bartenders											633	771	0.67	0.27
25 to 34 years	359	3.3	37.0	0.6	1.9	6.1	9.7	6.1	3.3	4.4				
35 to 44 years	442	0.9	29.4	2.0	1.4	8.8	9.7	9.5	2.7	4.7				
45 to 53 years	203	....	17.7	4.9	3.9	8.4	14.8	12.3	3.0	6.4				
55 to 64 years	52	....	1.9	5.8	9.6	21.2	9.6	13.5	1.9	....				
Basket makers											947	....	1.10	
Blacksmiths											1002	....	0.54	
25 to 34 years	216	4.2	29.6	1.9	1.4	6.5	7.4	5.6	3.7	10.2				
35 to 44 years	280	3.9	27.1	3.2	2.5	6.4	10.0	5.0	2.5	13.2				
45 to 54 years	409	1.7	15.9	9.0	7.1	10.8	8.1	8.8	3.2	8.3				
55 to 64 years	485	0.4	6.0	10.5	10.5	15.3	8.7	10.7	2.3	4.1				
Bleachers, dyers, laundry workers											979	966	0.79	0.58
Boat builders and carpenters											877	....	0.69	
Boatmen and sailors											1278	....	1.46	
25 to 34 years	242	6.6	16.5	1.7	0.8	4.1	6.2	4.5	3.3	31.8				
35 to 44 years	242	3.7	15.7	2.5	4.5	9.1	8.3	4.5	4.1	25.2				
45 to 54 years	309	0.6	12.0	6.5	4.9	10.7	9.4	9.4	1.0	22.7				
55 to 64 years	288	0.3	5.9	9.4	9.7	12.2	9.4	11.8	1.7	12.1				
Bookkeepers and accountants											511	560	0.75	0.24
25 to 34 years	347	5.5	42.9	1.2	1.4	5.5	5.5	2.9	5.2	4.3				
35 to 44 years	294	3.4	31.6	3.4	1.4	11.9	7.5	6.5	5.1	3.4				
45 to 54 years	280	1.4	12.9	8.2	6.4	13.2	9.3	10.7	4.6	3.6				
55 to 64 years	305	0.3	5.2	9.5	11.1	17.4	6.2	13.4	2.3	2.6				
Bookbinders											904	1238	0.69	0.63
Boot and shoe makers and repairers											642	932	0.68	0.41
25 to 34 years	256	4.7	35.5	1.2	1.2	7.0	7.4	4.3	3.9	8.2				
35 to 44 years	260	1.5	31.9	3.1	3.1	9.2	9.6	5.4	5.4	6.5				
45 to 54 years	339	0.6	16.8	4.4	7.1	10.0	13.0	14.7	5.3	3.2				
55 to 64 years	456	0.7	6.6	7.0	12.5	14.7	5.7	15.1	2.6	5.0				
Brass and aluminum workers											761	....	0.50	
Brewers and malsters											1106	....	1.16	
Brick and terra cotta workers											1118	....	0.89	
Bronze factory workers											890	....	0.23	
Brush and hair workers											910	....	0.95	

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Butchers	....	....	....	....	....	....	....	....	....	....	577	....	0.36	
25 to 34 years	208	3.4	39.4	1.4	1.9	5.3	10.1	5.8	2.9	8.7				
35 to 44 years	293	4.1	22.9	4.8	2.7	9.6	8.5	8.2	4.4	8.2				
45 to 54 years	341	1.8	13.8	6.7	4.1	13.2	5.6	10.3	5.3	6.5				
55 to 64 years	295	0.3	6.8	12.5	11.5	12.2	7.5	13.2	5.4	3.4				
Cabinet makers, wood polishers	....	....	....	....	....	....	....	....	....	....	810	....	0.72	
Cabmen and drivers	....	....	....	....	....	....	....	....	....	....	899	....	1.09	
Card-board and paper-box workers	....	....	....	....	....	....	....	....	....	....	1574	1165	1.06	0.49
Carpenters' helpers	....	....	....	....	....	....	....	....	....	....	1358	....	0.80	
Carpenters and joiners	....	....	....	....	....	....	....	....	....	....	877	....	0.68	
25 to 34 years	565	7.1	26.0	2.3	0.9	4.8	5.8	5.1	4.1	24.4				
35 to 44 years	874	3.3	22.5	4.8	3.3	7.7	8.7	5.9	3.8	15.8				
45 to 54 years	1256	2.1	13.6	7.1	6.5	10.4	8.9	8.9	3.8	12.0				
55 to 64 years	1621	0.7	8.5	10.0	10.0	16.3	7.7	9.3	3.2	7.3				
Cement mixers and hod carriers	....	....	....	....	....	....	....	....	....	....	1439	....	0.90	
Chemical industry workers	....	....	....	....	....	....	....	....	....	....	947	1342	0.85	0.85
Cigar and tobacco workers	....	....	....	....	....	....	....	....	....	....	909	1158	1.44	0.95
Clerks and salesmen and women	....	....	....	....	....	....	....	....	....	....	404	733	0.48	0.34
Clerks and copyists:														
25 to 34 years	1762	4.8	44.2	1.6	1.5	5.3	6.4	5.2	4.1	5.3				
35 to 44 years	1297	2.1	31.5	3.4	2.7	7.5	10.7	8.2	3.2	4.5				
45 to 54 years	1056	0.9	17.0	6.2	7.6	10.4	8.0	11.3	3.6	4.9				
55 to 64 years	848	0.8	7.2	8.0	10.0	15.4	7.5	15.6	1.7	3.4				
Clergymen:														
25 to 34 years	61	3.3	41.0	1.6	....	3.3	6.6	6.6	1.6	3.3				
35 to 44 years	104	5.8	19.2	5.8	7.7	9.6	7.7	7.7	1.0	3.8				
45 to 54 years	176	4.5	9.1	7.4	8.5	12.5	7.4	13.6	....	5.1				
55 to 64 years	255	1.2	2.7	9.4	10.6	14.5	7.1	17.3	0.4	4.3				
Compositors and type-setters	....	....	....	....	....	....	....	....	....	....	1155	....	0.83	
Concrete and asphalt workers	....	....	....	....	....	....	....	....	....	....	1320	....	0.71	
Confectioners	....	....	....	....	....	....	....	....	....	....	524	....	0.08	
Cooks	....	....	....	....	....	....	....	....	....	....	....	893	....	0.34

MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations: All causes	Per cent. of all causes at specified age: Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	No. of days of sickness per annum per 100 members[1]: Males	Females	No. of deaths per 100 members[1]: Males	Females
Coopers	570	....	....	....	....	....	....	....	....	....	1122	....	1.30	....
25 to 34 years	30	....	33.3	....	....	3.3	16.7	13.3	3.3	6.6				
35 to 44 years	59	....	45.8	1.7	....	6.8	10.2	5.1	5.1	10.2				
45 to 54 years	84	....	23.8	7.1	3.6	15.8	10.7	7.1	3.6	8.3				
55 to 64 years	89	1.1	10.1	14.6	6.7	16.9	9.0	7.9	2.2	3.4				
Decorators and painters:	....	....	....	....	....	....	....	....	....	....	1017	....	0.72	....
Domestic and personal service: males	....	....	....	....	....	....	....	....	....	....	....	....	....	....
25 to 34 years	7303	4.5	32.3	0.9	1.2	5.1	9.5	4.6	3.5	12.6				
35 to 44 years	8370	2.3	25.5	2.5	2.6	8.3	11.1	7.2	2.6	10.8				
45 to 54 years	8398	1.1	17.1	5.6	5.3	12.2	10.7	10.1	3.0	8.5				
55 to 64 years	6914	0.6	10.2	8.2	7.9	14.7	10.9	13.4	2.1	5.6				
Domestic and personal service: females	....	....	....	....	....	....	....	....	....	....	....	774	....	0.48
25 to 34 years	3155	3.2	33.2	3.0	1.5	6.0	6.5	4.7	2.1	3.0				
35 to 44 years	3066	2.2	21.3	9.2	3.9	10.4	6.6	7.8	1.1	2.1				
45 to 54 years	3232	1.1	10.1	14.1	8.9	13.1	8.2	10.7	0.8	2.7				
55 to 64 years	3280	0.6	5.0	13.9	12.8	15.2	8.5	12.2	0.8	2.5				
Janitors and sextons:														
25 to 34 years	78	....	47.4	2.6	....	....	10.3	10.3	1.3	3.8				
35 to 44 years	157	1.9	21.7	2.5	3.2	8.3	13.4	8.3	5.7	9.6				
45 to 54 years	219	0.9	14.6	9.1	6.8	13.7	11.9	10.0	5.0	5.5				
55 to 64 years	276	....	9.4	9.1	8.0	16.7	12.3	13.0	2.9	5.4				
Draymen, hackmen, teamsters, etc.	....	....	....	....	....	....	....	....	....	....	899	....	1.09	
25 to 34 years	1333	3.9	35.8	1.0	1.2	5.9	10.0	4.2	2.9	14.2				
35 to 44 years	1369	1.3	29.3	2.6	2.5	8.4	10.8	6.3	2.2	15.1				
45 to 54 years	1086	1.4	18.2	5.6	4.2	11.0	9.7	9.9	2.7	13.4				
55 to 64 years	736	0.5	10.2	9.9	10.5	14.1	8.7	12.2	1.5	12.0				
Dressmakers	....	....	....	....	....	....	....	....	....	....	....	933	....	0.50
25 to 34 years	186	3.2	40.3	1.6	1.1	5.9	5.4	5.4	2.7	3.8				
35 to 44 years	191	1.6	19.9	14.1	2.6	9.9	5.2	9.4	3.1	2.1				
45 to 54 years	195	....	11.8	24.6	6.7	11.8	8.2	5.6	1.0	3.1				
55 to 64 years	143	....	8.4	22.4	11.9	18.2	9.8	10.5	0.7	2.1				
Dyers, bleachers, and laundry workers	....	....	....	....	....	....	....	....	....	....	979	966	0.79	0.58

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No of days of sickness per annum per 100 members[1]		No of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age: Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Electric apparatus factory workers											722	....	0.60	
Engineers and firemen (not locomotive)											767	....	0.98	
25 to 34 years	471	8.3	25.7	1.5	0.2	6.6	6.2	3.2	3.6	18.9				
35 to 44 years	605	3.6	18.7	5.6	2.3	8.3	9.6	7.4	3.1	17.5				
45 to 54 years	694	0.9	10.5	7.9	6.1	12.2	9.4	10.4	4.5	9.9				
55 to 64 years	683	1.0	7.0	10.8	11.9	14.8	7.0	11.7	1.6	6.9				
Errand boys and girls, porters, etc.											621	1043	0.77	0.57
Excavation laborers and house wreckers											1519	....	1.03	
File makers											1117	....	1.13	
Flour mill workers											651	....	0.67	
Forgemen and blacksmiths											1002	....	0.54	
Furriers and fur dyers											736	1193	0.94	0.61
Gas workers											1084	....	0.66	
Glaziers											804	....	0.57	
Hair and brush workers											910	....	0.95	
Hatters											1124	....	1.15	
Hide industry workers											1123	1978	1.92	0.75
Hod carriers											1439	....	0.90	
Hotel kitchen helpers											689	1810	0.59	0.40
House wreckers											1519	....	1.03	
Illuminators photoengravers and photographers											573	814	0.77	0.41
Instruments of precision makers											955	1147	0.78	0.51
Iron founders and machinists											1189	1666	0.71	2.30
Jewelry industry, gold and silver											461	....	0.83	
Iron and steel workers											1256	....	0.69	
25 to 34 years	524	5.0	25.2	2.3	1.1	3.6	10.9	3.4	3.6	28.1				
35 to 44 years	617	3.6	22.0	2.3	2.8	7.9	12.2	5.5	2.8	20.9				
45 to 54 years	556	1.3	15.3	4.0	6.8	10.3	11.3	7.9	2.9	14.0				
55 to 64 years	394	1.0	7.9	10.2	8.6	14.7	11.2	8.4	1.5	7.1				
Laborers (not specified)														
25 to 34 years	5,180	5.1	30.8	1.0	1.2	5.0	10.2	4.5	2.9	13.9				
35 to 44 years	5,606	2.5	25.9	2.4	2.7	8.0	12.1	6.4	1.7	12.6				
45 to 54 years	5,635	1.1	18.2	5.5	4.8	12.5	11.9	9.1	2.5	9.3				
55 to 64 years	4,797	0.7	10.7	8.2	7.4	14.1	12.2	13.5	1.8	5.7				

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations: All causes	Per cent. of all causes at specified age: Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	No. of days of sickness per annum per 100 members[1]: Males	Females	No. of deaths per 100 members[1]: Males	Females
Lace factory workers											620	910	0.35	0.54
Ladies' tailors and milliners											691	847	0.82	0.55
Laundresses											....	966	....	0.58
25 to 34 years	212	3.3	33.0	3.8	1.4	7.5	4.2	3.8	1.9	4.7				
35 to 44 years	216	3.2	18.5	7.9	3.2	12.0	8.8	9.3	1.9	2.8				
45 to 54 years	243	1.6	11.5	8.6	8.2	15.2	11.5	9.1	0.4	1.6				
55 to 64 years	165	....	2.4	10.3	12.7	13.9	10.3	13.9	....	3.0				
Lawyers:														
25 to 34 years	105	8.6	25.7	1.9	1.9	7.6	1.9	....	7.6	9.5				
35 to 44 years	141	2.1	19.9	2.1	4.3	7.1	17.7	14.9	4.3	3.5				
45 to 54 years	236	0.4	8.9	6.8	13.1	9.7	8.9	9.3	4.2	5.5				
55 to 64 years	303	0.3	5.0	8.6	12.5	16.5	6.6	12.5	1.7	2.6				
Leather, artificial and oil-cloth workers											878	....	1.38	
Locksmiths											892	....	0.59	
Lithographers											650	....	0.52	
Machinists											1189	1666	0.71	2.30
25 to 34 years	574	5.4	35.0	1.2	1.7	4.5	5.9	4.0	5.1	15.5				
35 to 44 years	567	3.5	23.8	3.7	1.9	7.8	9.2	4.4	4.4	11.6				
45 to 54 years	567	0.7	13.1	8.1	4.2	13.9	10.6	11.3	4.4	8.6				
55 to 64 years	556	1.1	7.2	7.7	12.8	16.2	8.8	12.9	2.3	5.6				
Manufacturers and officials, etc:														
25 to 34 years	164	5.5	28.0	1.8	3.0	3.0	6.7	3.0	3.0	18.3				
35 to 44 years	327	4.3	14.1	6.4	4.3	8.3	8.3	8.3	1.8	14.4				
45 to 54 years	526	2.7	7.2	7.4	7.4	9.5	6.3	12.2	5.7	9.9				
55 to 64 years	708	1.1	3.2	5.6	11.2	15.7	6.6	11.7	1.1	5.9				
Manufacturing and mechanical pursuits, males:														
25 to 34 years	8,481	4.7	30.8	1.5	1.1	4.8	7.0	3.8	4.0	21.9				
35 to 44 years	10,405	2.5	25.0	3.6	2.6	7.2	9.1	6.5	3.6	16.7				
45 to 54 years	11,597	1.4	15.6	6.6	5.7	10.7	9.0	9.6	3.8	11.0				
55 to 64 years	11,408	0.6	8.2	9.3	10.1	15.0	8.0	11.5	2.6	6.3				
Manufacturing and mechanical pursuits, females:														
25 to 34 years	885	2.4	40.8	2.9	1.2	7.6	5.1	5.0	3.3	2.8				

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations: All causes	Per cent. of all causes at specified age: Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	No. of days of sickness per annum per 100 members[1]: Males	Females	No. of deaths per 100 members[1]: Males	Females
35 to 44 years	730	1.1	26.3	11.4	3.2	12.3	5.5	7.8	1.6	2.6				
45 to 54 years	600	0.3	12.3	20.3	7.0	12.3	7.7	8.0	0.7	3.2				
55 to 64 years	462	0.4	9.3	18.8	11.3	16.5	10.4	8.7	0.4	3.0				
Marble and stone cutters	822	....	....	....	....	....	....	....	....	....	1294	....	1.27	
25 to 34 years	75	2.7	46.7	....	1.3	6.7	12.2	5.3	1.3	10.7				
35 to 44 years	156	2.6	41.0	3.8	1.9	3.2	6.8	5.1	....	10.3				
45 to 54 years	177	0.6	42.9	5.1	4.0	8.5	6.9	8.5	0.6	5.6				
55 to 64 years	218	0.5	19.3	7.8	6.9	13.8	6.7	9.6	0.9	3.2				
Masons (brick and stone)	....	....	....	....	....	....	....	....	....	....	777	....	0.70	
25 to 34 years	188	4.8	33.0	1.1	1.6	3.7	10.1	3.2	2.7	17.0				
35 to 44 years	346	0.6	29.8	3.2	1.7	9.0	10.7	5.8	4.3	15.6				
45 to 54 years	419	1.4	19.6	6.7	4.5	11.9	8.6	7.9	4.5	11.7				
55 to 64 years	512	0.2	7.4	11.3	9.4	14.5	7.8	12.3	3.3	8.0				
Merchants and dealers (except wholesale):														
25 to 34 years	837	6.1	30.3	2.2	2.2	6.5	8.1	5.1	3.2	6.4				
35 to 44 years	1,369	3.0	18.7	4.4	4.3	9.4	7.8	9.8	4.1	5.8				
45 to 54 years	1,901	1.4	10.2	8.1	6.9	12.3	6.3	12.1	3.8	4.5				
55 to 64 years	2,091	0.9	5.2	10.2	11.8	14.8	6.6	13.3	1.8	2.2				
Metal polishers and grinders	....	....	....	....	....	....	....	....	....	....	1215	1456	1.02	0.81
Milliners	....	....	....	....	....	....	....	....	....	....	....	847	....	0.55
Miners and quarrymen	....	....	....	....	....	....	....	....	....	....	1496	....	0.88	
25 to 34 years	1,132	3.7	8.4	1.1	....	2.1	5.5	1.5	2.0	61.3				
35 to 44 years	1,135	2.4	10.5	2.0	1.9	3.1	9.4	3.1	1.9	47.0				
45 to 54 years	993	1.4	11.3	4.6	4.3	8.0	10.7	4.1	1.6	27.3				
55 to 64 years	725	0.3	10.8	7.6	6.9	15.6	11.4	6.2	1.2	13.9				
Miners, coal:*														
25 to 34 years	151	....	11.9	1.5	0.7	4.4	7.4	1.5	2.2	43.7				
35 to 44 years	182	....	12.1	2.7	1.6	6.6	10.4	6.6	2.2	30.2				
45 to 54 years	319	....	6.6	3.4	4.1	8.8	14.7	7.8	2.2	15.7				
55 to 64 years	407	....	3.4	6.9	7.6	10.8	11.3	11.5	0.7	9.3				
Oils, essential and volatile workers	....	....	....	....	....	....	....	....	....	....	778	....	0.69	
Oil-cloth and artificial leather makers	....	....	....	....	....	....	....	....	....	....	878	....	1.38	

* Compiled from mortality statistics of the Metropolitan Life Insurance Company, New York, published September, 1915.

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations: All causes	Per cent. of all causes at specified age: Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	No. of days of sickness per annum per 100 members[1]: Males	Females	No. of deaths per 100 members[1]: Males	Females
Opticians, clock and watch makers											674	....	0.66	
Painters and decorators											1017	....	0.72	
Painters, glaziers and varnishers											1017	....	0.72	
25 to 34 years	471	3.2	37.2	1.7	0.4	4.5	6.8	5.5	4.0	16.8				
35 to 44 years	756	0.8	28.4	3.4	4.1	7.3	8.7	11.2	2.4	13.0				
45 to 54 years	842	0.7	17.3	5.1	6.9	9.7	7.8	13.4	3.3	11.0				
55 to 64 years	733	0.4	9.8	7.5	11.1	13.5	7.6	16.2	2.5	5.9				
Paper-box factory workers											1574	....	1.06	
Paper-goods factory workers											827	1202	0.96	0.57
Pavers and asphalters											1317	....	0.88	
Physicians and surgeons:														
25 to 34 years	99	4.0	21.2	2.0	4.0	6.1	12.1	4.0	7.1	11.1				
35 to 44 years	184	3.8	15.8	2.2	3.3	5.4	12.0	6.0	2.7	6.5				
45 to 54 years	251	2.0	9.2	5.2	7.2	7.6	8.0	16.7	3.6	5.6				
55 to 64 years	301	0.7	4.0	11.3	10.0	15.0	4.3	10.6	2.7	3.7				
Piano and muscial instrument makers											845	1055	0.88	0.55
Picture-frame factory workers											815	1214	0.81	0.15
Plasterers											738	....	0.46	
Plate printers											718	....	0.52	
Plumbers, and gas and steam fitters											836	....	0.61	
25 to 34 years	271	4.8	43.2	1.5	2.6	5.5	7.4	4.8	1.5	11.4				
35 to 44 years	323	2.5	35.9	2.8	1.5	6.8	11.1	6.8	3.1	10.5				
45 to 54 years	213	1.9	14.6	5.2	8.0	12.2	12.7	6.6	3.3	11.3				
55 to 64 years	131	....	18.3	6.9	10.7	17.6	9.2	6.9	....	3.8				
Porters and helpers (in stores, etc.)														
25 to 34 years	264	1.5	38.3	0.4	0.8	6.1	10.2	3.8	1.5	6.4				
35 to 44 years	323	1.2	31.6	1.9	3.4	11.5	11.5	6.8	2.2	5.6				
45 to 54 years	264	1.5	21.6	4.5	1.9	16.3	15.2	16.7	1.9	4.5				
55 to 64 years	155	0.6	10.3	6.5	7.7	14.8	9.7	14.8	1.3	11.0				
Potters and pottery workers											957	....	0.77	

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Printers, lithographers, and pressmen	......	....	....	....	....	....	....	....	....	....	851	1130	0.58	0.5[illegible]
25 to 34 years	291	2.7	51.5	0.7	2.7	4.1	5.5	3.4	4.1	4.1				
35 to 44 years	328	2.4	32.6	5.5	3.7	8.2	7.6	7.3	5.2	7.3				
45 to 54 years	265	1.9	22.3	7.2	5.7	9.4	8.3	10.2	1.5	4.5				
55 to 64 years	168	0.6	6.5	9.5	11.3	14.9	9.5	14.3	5.4	4.2				
Professional service, males:														
25 to 34 years	1192	5.7	33.6	1.4	1.3	4.9	5.7	3.3	4.6	14.4				
35 to 44 years	1329	2.9	20.4	3.2	4.5	7.2	9.9	7.8	4.7	7.7				
45 to 54 years	1567	1.9	9.9	6.3	8.1	10.8	7.8	12.3	3.6	6.2				
55 to 64 years	1712	0.7	4.4	9.5	10.8	15.7	6.9	13.0	1.8	3.9				
Professional service, females:														
25 to 34 years	385	5.5	37.1	4.7	1.3	6.8	4.7	2.9	1.3	3.6				
35 to 44 years	293	2.0	16.0	15.7	6.1	8.5	9.2	6.8	....	3.4				
45 to 54 years	268	0.7	7.5	20.9	7.5	9.7	5.2	9.0	1.9	3.7				
55 to 64 years	196	0.5	7.7	15.8	10.2	11.7	8.7	10.7	0.5	2.0				
Quarrymen and stone-breakers	......	....	....	....	....	....	....	....	....	....	1496	....	0.88	
Rubber goods workers	......	....	....	....	....	....	....	....	....	....	1028	1494	0.90	0.83
Refiners in sugar factories	......	....	....	....	....	....	....	....	....	....	1443	....	0.54	
Railway enginemen and trainmen*	947													
25 to 34 years	......	3.3	14.7	0.7	0.3	1.3	4.7	2.0	2.3	56.0				
35 to 44 years	......	3.5	24.9	1.7	1.2	5.2	6.9	7.5	2.9	28.3				
45 to 54 years	......	....	13.8	0.9	6.4	10.1	9.2	9.2	2.8	18.3				
55 to 64 years	......	....	7.4	5.3	13.8	13.8	6.4	10.6	2.1	16.0				
Railroad street employees	......	....	....	....	....	....	....	....	....	....	870	....	0.76	
Saddlers, leather workers	......	....	....	....	....	....	....	....	....	....	684	....	0.49	
Sailors	......	....	....	....	....	....	....	....	....	....	1278	....	1.46	
Saw mill workers	......	....	....	....	....	....	....	....	....	....	1179	....	0.73	
Shoemakers, see boots and shoemaker	......	....	....	....	....	....	....	....	....	....	642	....	0.68	

* Compiled from mortality statistics of the Metropolitan Life Insurance Company, New York. September, 1915.

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MORTALITY AND MORBIDITY STATISTICS.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Steam-railroad employees:														
25 to 34 years	1382	2.7	10.5	0.7	0.9	1.7	2.6	1.5	1.2	67.9				
35 to 44 years	1133	1.9	9.2	1.9	1.2	3.9	4.4	2.7	2.5	56.3				
45 to 54 years	941	1.2	6.6	5.3	3.5	5.8	5.1	6.3	1.3	47.7				
55 to 64 years	724	....	3.2	8.1	7.9	12.2	5.7	9.1	1.0	30.2				
Servants and waiters											633	771	0.67	0.27
25 to 34 years	693	2.9	40.4	0.6	1.3	6.3	7.4	4.2	5.5	7.8				
35 to 44 years	701	1.7	29.7	2.3	1.4	11.0	7.8	8.4	3.9	6.7				
45 to 54 years	644	0.8	20.2	4.5	6.7	14.6	7.5	12.7	2.6	6.8				
55 to 64 years	407	0.2	17.0	9.6	8.1	11.8	9.6	12.3	2.5	2.9				
Servants and waiters, females:														
25 to 34 years	2704	2.9	33.9	3.0	1.5	5.9	6.6	4.7	2.0	2.8				
35 to 44 years	2578	1.8	22.0	9.3	3.9	10.1	6.2	7.7	0.8	1.9				
45 to 54 years	2671	1.0	10.2	14.4	9.1	12.9	7.9	11.3	0.7	2.6				
55 to 64 years	2760	0.5	5.5	13.6	13.0	15.7	8.4	12.2	0.6	2.3				
Storage battery workers											722	1276	0.60	0.55
Tailors											715	884	0.88	0.56
25 to 34 years	326	2.1	37.7	2.1	1.5	6.1	7.4	6.4	6.1	7.4				
35 to 44 years	415	2.7	33.7	4.3	1.7	8.0	5.8	10.1	6.3	3.4				
45 to 54 years	456	0.9	16.7	8.1	6.1	9.9	8.1	10.1	4.6	4.8				
55 to 64 years	361	....	6.9	13.6	8.3	13.6	9.4	12.2	4.7	3.6				
Tallow and soap factory workers											....	1035	....	1.14
Tanners and leather dyers											1049	....	0.46	....
Teachers and professors in colleges, etc., females:														
25 to 34 years	269	6.3	38.7	4.8	1.5	5.2	5.2	2.6	0.7	3.3				
35 to 44 years	194	3.1	19.1	13.9	4.1	9.3	11.9	7.2	....	1.5				
45 to 54 years	167	1.2	7.8	22.2	7.8	8.4	7.2	9.0	1.2	3.6				
55 to 64 years	130	0.8	7.7	16.2	6.9	10.0	11.5	10.0	....	2.3				
Terra cotta and brick workers											1118	....	0.89	
Textile mill workers*	2390										876	1229	0.83	0.58
25 to 34 years		5.3	47.5	0.4	0.4	5.7	4.3	2.5	1.4	5.7				
35 to 44 years		1.3	37.7	1.3	1.0	5.5	9.0	7.1	1.0	6.8				
45 to 54 years		1.5	18.0	4.8	5.8	10.3	6.3	12.5	3.5	4.5				
55 to 64 years		0.8	8.9	7.1	9.9	17.0	5.7	12.1	1.4	3.4				

* Compiled from mortality statistics of the Metropolitan Life Insurance Company, New York, published September, 1915.

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Mortality and Morbidity Statistics.—*Continued*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia (all forms)	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Tin plate and tinware makers	681	....	....	....	....	....	....	....	....	....	824	....	0.79	
25 to 34 years	92	7.6	37.0	1.1	....	8.7	5.4	4.3	5.4	15.2				
35 to 44 years	120	....	32.5	3.3	1.7	6.7	8.3	5.8	1.7	13.3				
45 to 54 years	134	0.7	17.9	7.5	6.7	13.4	9.0	9.0	3.7	9.7				
55 to 64 years	136	....	7.4	9.6	5.9	15.4	7.4	13.2	2.9	6.6				
Tobacco and cigar factory workers	982	....	....	....	....	....	....	....	....	....	909	1158	1.44	0.95
25 to 34 years	136	5.1	49.3	0.7	....	2.9	6.6	4.4	5.1	5.1				
35 to 44 years	181	....	33.7	1.1	4.4	12.2	6.1	3.3	4.4	7.7				
45 to 54 years	233	....	23.2	9.4	3.0	11.2	6.9	9.0	5.2	5.6				
55 to 64 years	187	0.5	8.0	12.3	7.0	11.8	4.8	19.3	4.3	2.7				
Typefounders, stereotypers	....	....	....	....	....	....	....	....	....	....	1064	1108	(?)	0.89
Typesetters and compositors	....	....	....	....	....	....	....	....	....	....	1155	....	0.83	(?)
Trade and transportation, males:														
25 to 34 years	7948	4.4	31.9	1.4	1.2	4.7	6.5	4.2	3.3	20.5				
35 to 44 years	8219	2.4	22.8	3.3	2.8	8.0	8.7	6.8	3.9	15.8				
45 to 54 years	8333	1.3	12.3	7.1	6.2	10.8	7.8	10.9	3.4	12.3				
55 to 64 years	7495	0.6	5.8	9.6	10.5	15.0	7.3	13.2	2.0	7.5				
Trade and transportation, females:														
25 to 34 years	667	4.3	39.9	3.0	0.9	6.4	3.6	5.2	3.1	3.0				
35 to 44 years	348	1.4	16.1	11.5	4.0	10.1	4.9	8.0	1.7	5.2				
45 to 54 years	207	1.4	7.7	18.8	6.8	8.7	9.2	9.2	0.5	2.4				
55 to 64 years	121	0.8	2.5	14.9	9.1	19.0	17.4	8.3	1.7	1.6				
Upholsterers	....	....	....	....	....	....	....	....	....	....	720	....	0.69	
Veneer factory workers	....	....	....	....	....	....	....	....	....	....	1154	....	0.88	
Watchmen, policemen, firemen, etc.:														
25 to 34 years	190	2.1	24.2	1.6	2.1	4.2	6.8	5.8	4.7	21.1				
35 to 44 years	315	2.5	17.8	1.9	2.5	9.2	8.3	6.7	6.0	14.6				
45 to 54 years	527	2.1	9.5	8.2	6.8	15.6	6.8	11.4	4.7	11.2				
55 to 64 years	657	0.3	4.9	7.5	9.9	18.2	7.8	12.3	3.5	8.4				
Waiters	....	....	....	....	....	....	....	....	....	....	633	771	0.67	0.27
Weaving mill operatives	....	....	....	....	....	....	....	....	....	....	557	....	0.75	
Wicker ware and basket workers	....	....	....	....	....	....	....	....	....	....	947	....	1.10	

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MORTALITY AND MORBIDITY STATISTICS.—*Concluded*

Occupation and age at death	Deaths of persons at least 10 years of age engaged in certain specified occupations										No. of days of sickness per annum per 100 members[1]		No. of deaths per 100 members[1]	
	All causes	Per cent. of all causes at specified age												
		Typhoid fever	Tuberculosis of lungs	Cancer	Apoplexy and paralysis	Heart disease	Pneumonia all forms	Bright's disease	Suicide	Accident	Males	Females	Males	Females
Woodworkers:														
25 to 34 years	197	2.0	40.6	1.0	1.0	3.6	7.6	2.5	6.6	18.3				
35 to 44 years	201	2.5	25.4	3.5	3.0	10.0	7.0	8.5	7.0	17.4				
45 to 54 years	264	1.1	18.6	7.2	4.2	10.2	8.7	10.6	3.4	12.9				
55 to 64 years	248	0.4	11.3	9.7	10.9	16.5	4.4	10.9	3.6	6.0				
Yeast factory workers	......	....	....	....	....	....	....	....	....	....	863	....	0.48	
Other miscellaneous industries:														
25 to 34 years	571	5.4	30.6	1.2	1.6	5.8	7.7	2.3	4.2	21.9				
35 to 44 years	566	2.5	23.3	3.0	3.0	6.2	10.6	7.2	3.2	16.1				
45 to 54 years	604	1.7	13.6	6.3	6.8	8.9	10.4	9.9	4.0	12.7				
55 to 64 years	523	0.2	7.8	9.6	10.5	16.8	6.9	8.8	2.5	6.1				

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PART III

THE RELATION OF HOSPITAL AND MEDICAL SCHOOL WORK, STATISTICS, GOVERNMENT STUDY AND LEGISLATION TO OCCUPATIONAL DISEASE

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DIVISION I

The Function of Clinics in the Prevention of Occupational Diseases

CHAPTER I

THE MILAN CLINIC FOR OCCUPATIONAL DISEASES. ITS ORIGIN, PURPOSES AND ACTIVITIES

BY PROF. DR. L. DEVOTO, Milan, Italy

The Milan Clinic for Occupational Diseases is unique at present in the scientific world, and marks a period of great historic importance in modern Italian life.

Since the very first days of the reign of Victor Emanuel III, public opinion has been interested in vital hygienic and social problems. Much has been said on malaria and the Roman waste-lands; on pellagra and the work in the rice fields and in the mines; on woman's and children's work; on Government inspection of labor; on accidents in general; and on assistance to invalids and to the aged poor.

The discussions were so wide and conclusive as to gain, between the years 1901 and 1902, the approval by Parliament of a series of social laws intended to remedy those painful conditions.

It was in that period that the city of Milan elaborated comprehensive regulations imposing on the city factories those strict hygienic rules that later were highly commended by the Government, and by the administration of other cities. Then followed the foundation of several institutions in Milan, which afforded physicians the opportunity of perfecting and specializing their knowledge; among these is the Clinic for Occupational Diseases.

The purposes of the Clinic are as follows: To study scientifically the causes of occupational diseases and to spread its clinical knowledge among physicians; to gather in the Clinic all workmen apparently or decidedly affected by occupational diseases, whether in incipient or advanced stage, for the purposes of diagnostical and therapeutical experiments, and to examine systematically the health conditions of workmen engaged in industries of all kinds, and especially those working in unhygienic occupations.

After two years of political vicissitudes the Italian Parliament passed a law recognizing the Industrial Clinic Institutions as "Post-University" and authorizing them to award diplomas.

In the year 1906 the First International Congress of Occupational Diseases was held in Milan and was a great success. This Congress, appreciating the vast importance of the industrial clinic-hygiene problems, suggested that the city secure larger ground for the erection of the Clinic than was intended and

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more means to provide it with a stronger scientific organization, which caused another delay.

Finally in the month of December, 1907, the laying of the corner-stone took place, and the building, completely finished, was inaugurated March 20, 1910.

The administration of the Institutes was entrusted to a Council of Members.

Calling me to the Directorship of the Clinic in 1906,* the city of Milan gave me power to enter agreements for the construction and disposition of the building which has since been erected in an area of 5000 square meters, actually covering a surface of 1500 square meters in the center of the hospital district of Milan, and not far from the leading labor establishments.

The Clinic consists of three buildings, which include:

Five dispensary rooms, also used for consultation.

Eight infirmaries and six isolating rooms of the capacity of 110 beds with the necessary smaller rooms used for first analysis.

Three physiopathological laboratories and one room for the study of experimental fatigue.

Three bacteriological laboratories.

Five chemical laboratories.

Five pathological, histological and microscopical laboratories.

One auditorium for 200 physicians and three demonstration rooms.

One large library.

One archives room.

Three Röntgenological laboratories.

Five rooms for the Professional Hygiene and Pathology Museum.

One refrigerator room.

One anatomic museum room.

One autopsy room.

The idea of having the autopsy and anatomic museum rooms in the same institute is worthy of the highest praise; it makes research easier and enables physicians taking the courses or working in the Clinic not only to watch the autopsy, but also to follow all the further investigations required for the anatomical diagnosis.

The laboratories have proved to be of great value to the Clinic not only for clinical study but also for experimental and anatomopathological studies. It is now more and more realized that it would have been useless to establish a Clinic which represents a departure from trodden paths, and which faces, besides its difficult problems, a considerable amount of public diffidence, without furnishing it with an abundance of scientific material. Better not

* When in the year 1901 I was a Professor of Internal Pathology at the University of Pavia, I initiated a clinical course on Occupational Diseases which proved very successful, and started the publication of a review of Industrial Physiology, Pathology and Hygiene, by the title: "Il Lavoro" ("Work") I left the University of Pavia in 1906 when the opening of the Clinic was approaching.

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do anything than bring into existence an ill-appointed institution, especially in a city like Milan, where neither a Faculty nor clinical facilities existed, and where there were no laboratories available.

Without counting the services kindly rendered by city officials, engineers, assistants and by all of the civil service workshops, the Clinic has cost over 1,000,000 lires, though the estimated expenditure called for a much smaller sum.

As for me, who took upon myself a part of the responsibility for this excessive expenditure in the building of the Clinic, I feel that, if this attitude of mine has been wrong, there is but one way for me to make reparation, and that is to do all I can to make the Clinic as useful as possible to the workers of Milan.

As things are now, only one-half of the 110 beds that the Clinic provides can be occupied, but I believe that we will have all the beds of the Clinic in use as soon as the Compulsory Insurance Act becomes a law in Italy, as it has in other countries.*

Some of the Members of the Faculty were my colleagues at the University of Pavia, some were assistants in other Italian or foreign universities, and all have had ample opportunity to deepen their studies in the particular branches that they had chosen and in which they were called here to lead and to teach or assist the 36 young physicians for whom there are positions.

Ever since the opening of the Clinic I deemed it necessary to establish a special and active propaganda of hygiene among the working classes. The results obtained are indeed remarkable.

The work is conducted in the following manner:

1. By clinical teaching to physicians.
2. By the treatment of the patients admitted in the Clinic.
3. By scientific work in the field of occupational pathology.
4. By consultations with and examinations of workmen; also by exercising a general control of their health and conditions.
5. By inspections and investigations.
6. By a popular propaganda of occupational hygiene.

Rather than stop to say more in a general way on these activities, I will relate how they have been applied in these first few years of the existence of the Clinic.

I. DIDACTIC ACTIVITY OF THE CLINIC

For the Clinic to undertake the task of teaching physicians the treatment and prevention of diseases such as the so-called *saturnism*, *mercurialism*, *anilinism*, *phosphorism*, etc., and of all other occupational intoxications, including *professional infections* (anchylostomiasis, carbuncles, etc.) the *neurosis* due to mechanical work, *caissonism*, *pneumokoniosis*, and miner's *phthisis*, is only natural. But a Clinic establishment for the purpose of diminishing the antiphysiologic factors of labor, for instructing physicians in that line, and

*A compulsory accident insurance law was passed in 1898 and amended in 1903. A compulsory maternity insurance law was passed in 1910. A voluntary sickness insurance and for old age insurance exist, the latter is compulsory for some of the government employees, but so far no compulsory health insurance system has been adopted in Italy.

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for awarding diplomas has another field of action, which is less definite, as yet, but much more important. It is the valuation of the occupational factor, often concealed, which must be more deeply considered. When this is discovered, then the steps for prevention can be more easily taken. Here are some of the points which are frequently used as clinical illustrations and which lead us to affirm strongly:

(*a*) That the relation between continual effort and alteration of the heart and of the blood-vessels is closer than generally believed.

(*b*) That healthy persons who undertake hard work, without having the adequate physical resistance, are bound to suffer alteration or displacement of some organs.

(*c*) That some visceral localizations of syphilis are related to abuse of strength; to local traumas.

(*d*) That hard labor may hinder, in workmen who have recently recovered from infective diseases, the restitution of normal functions of those organs which were involved by the infection (myocardium, lungs).

(*e*) That a loose technical appreciation of the health conditions of boys and girls licensed to work, may cause serious disturbances.

(*f*) That a wholesome occupation may still cause disturbances if the environment is unhygienic (dampness, poor light, insufficient air).

(*g*) That work requiring strained positions must be done in short periods, alternated by exercise.

(*h*) That occupational infantilism occurs oftener than it is believed.

(*i*) That labor is to be considered as a factor capable of forming special clinical pictures.

Legislators may distinguish between diseases arising undoubtedly from work through poison and those due to the diffusion of bacteria, or those caused by intermittent or contingent factors, but we, as physicians who must look at diseases especially from the viewpoint of their cure, and above all of their prevention, and who live outside the pale of mere speculation, have no doubt that, admitting the necessity of an institute for this specific study and teaching and for the prophylaxis of labor diseases, it is the task of such a Clinical Institute and of its Staff to make researches for and seek the demonstration of the agent creative of the diseases, inasmuch as it is centered in antiphysiological and antihygienic work.

I am also hopeful that, little by little, the Faculty of the University Medical Clinics will come to see the necessity of helping their students to look at diseases from the point of view of the social influence, and to give them, besides the ordinary clinical lessons, those lessons that have far-reaching social import.

The young physician and the student, if taught to analyze, with an objective, unbiased criticism, the causes contributing to the pathogenic forces bad surroundings or unhygienical work, will certainly become a good clinic physician as well as a good social worker.

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The official lessons of the Director of the Clinic are public. They begin November 1, and end June 15. During the fall vacations, and also during the school year, rapid courses of professional pathology and hygiene, of tuberculosis, pellagra, diagnosis, and the use of X-rays, are given, with the assistance of Professors attached to the Clinic.

The fees range from 40 lires (without laboratory) to 480 (including laboratory) a year.

To obtain a diploma, the student must have attended all the courses of the Clinic for no less than 12 months, write an original thesis on an argument in harmony with the ultimate purposes of the Clinic, and pass a practical clinic examination, and a theoretical test as well.

The graduates, up to now, number ten, and ten others are preparing their theses. The number of physicians who have attended the lessons and who have done work in the laboratories and dispensaries is 380.

Out of the funds donated to the Clinic, it is possible for the Administration to award small scholarships to those civil service doctors who wish to complete, by clinical observation and laboratory research, the study of the cases of professional pathology which they meet in their daily practice.

2. ASSISTANCE TO PATIENTS

Workmen affected by direct or contributory causes arising from their occupations are taken in the Clinic: (*a*) to be treated; (*b*) to establish a diagnosis, when it is not possible to do so in the dispensary; (*c*) for teaching purposes (and I wish to say here that our working people willingly submit to such examinations when requested) although, whenever it is possible, we prefer out-patients.

The Clinic rooms are very well aired, ventilated, heated and dusted (vacuum cleaner), and every bed has about 1200 cu. ft. of air-space; every ward has its own analysis room.

It is superfluous for me to enter into the details of the different methods of treatment, for, as may be easily understood, they vary so, some being of a pharmacologic order, some of a physical order, etc., but I wish to say this, that all patients and their relatives are provided with a detailed account of the disease, and every person who is discharged from the Clinic, besides the better health that it has been possible to give him, also has the privilege of taking with him a good knowledge of hygienic rules which he may profitably impart to his fellow-workers.

As a general rule, people affected by pulmonary tuberculosis are not admitted. Only for special didactic purposes are patients affected by pneumokoniosis and eventual tubercular processes admitted to the Clinic, and these are placed in the isolation rooms.

The most important dispensary cases which have presented themselves during the week are reviewed each Saturday and briefly discussed for the benefit of the physicians.

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The Clinic offers to its patients a large garden and several terraces. This is not the ideal. Italy, so far, has no convalescence or "Erholung," places as the Germans have it, and something of that sort is needed for a Clinic which treats persons affected by apical catarrh, poor blood, states of neurosis, alterations of the cardiovascular apparatus, paralysis, etc., but I hope that in the near future we may have some ideal climatic balneological station on the lake of Garda (90 minutes from Milan). The owner of the *Sirmione* Station has already offered the Clinic a large piece of ground for that purpose, and if the plan can be carried out, the Station will remain open all the year round. In that vicinity there is also a spring of chlorosulphuric water, something like that of Aix-La-Chapelle, which is very good for cases of lead intoxication. The delightful climate of the spot would make the Station all the more beneficial.

3. SCIENTIFIC PRODUCTION

The Clinic's scientific production consists of researches, the findings of which are made known widely, so as to obtain the views of all physicians and institutions working in other fields, as well as those of the city doctors. The patients coming to the dispensary or received in the Clinic frequently offer splendid material for special research. I will here give a short account of what has been done up to the present time and of the results attained.

Overexertion has been the object of many laboratory researches which are conducted as follows: *histopathologically* by *Cesa Bianchi*, *experimentally* by *Ciovini*, *immunitarily* by *Vallardi*, and *chemically* by *Preti;* all these led us to ascertain:

1. That work, even when very hard, does not produce immunitary reactions.

2. That hard work originates alternations of the cardiac muscular fibers which, if not extremely serious, may be repaired by rest and good nourishment, but which are otherwise irreparable. The suprarenal capsules are also subject to alteration by hard work.

3. That hard work generates a conspicuous neutrophilic polynucleosis connected with a transient disorder of the blood gases (excess of carbon dioxide) caused by a function of the leucocytes versus carbon dioxide.

4. That from hard muscular work there arises a production of acetone and also an increase of the antitryptic power of the blood serum, and that in another kind of muscular exertion, in the excitement of persons mentally affected, a remarkable elimination of uric acid is noted.

5. That periods of great strain in working people (harvest time) lead to prostration of the strength and pressure of the heart.

Dusts were made the object of special experimental studies by Prof. Cesa Bianchi and myself, and this is our point of view:

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(*a*) By causing animals to inhale dust we have produced only pulmonary tuberculosis, regardless of the way in which the tubercular germ is introduced, while without the use of dust general tubercular processes result.

(*b*) Dusty trades frequently produce acoustic reactions of the pulmonary apexes which look like, but are not, the result of a tubercular process. The germ of tuberculosis will later thrive because dusts have prepared the soil for it.

Chronic poisoning derived from *lead* inhalation has been studied from different points of view, and we are in a position to say that the first alterations come from disturbances of a chemical order (production of uric acid on account of prevented uricolysis or of uric reconstruction) and from alterations of cellular elements to which lead adheres more durably, both in its passage and during its elimination, and because of an attraction of the said cellular elements toward lead, whether alcohol, exertion, light, etc., are contributory or not, they prepare the way under the progressive march of intoxication, for vasal alterations and, with or without these, but aided by other circumstances, as, the habit of unusual effort, damp or antihygienic environment, insufficient or irrational nourishment, wine, liquors, tobacco, and common diseases, the condition progresses and we enter into the realm of real saturnism, that is, into that large, complete and complex range of phenomena which has caused lead to be called the poison of protoplasm on a large scale.

In studying pregnancy cases of varnishers' wives in the Clinic, it has been ascertained that the mortality of the descendants is 49.7 per cent., and the occurrence of abortions has been calculated as 10.3 per cent.

As to *phosphorism*, Dr. Vallardi, who had the opportunity to study about 50 cases of phosphoric necrosis, made experimental researches that lead us to state:

1. The pathognomonic manifestation of chronic phosphorism in the human body is shown by the necrosis of the jawbone.

2. The other symptomatical manifestations especially frequent among workmen who are always in contact with phosphorus have no specific characters.

3. It is not possible to reproduce in animals, poisoned chronically with phosphorus, a phenomenon resembling nearly enough that which appears in the necrosis of man's jawbone.

4. On the contrary, chronic poisoning of animals by phosphorus through the respiratory or the gastric channels or by both ways simultaneously produces modifications of the blood crasis, injuries of the glandular parenchyma, of a degenerative type (especially of the liver), and constant alterations of the material exchange.

None of these manifestations, considered by itself, can be regarded as specific.

The Clinic has had about 100 cases of *pellagra* in these past years, and

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from the study made of this disease we are led to admit that there is no pellagra without maize (corn).

In conformity with the views expressed by me years ago about the paresis appearing in cases of pellagra in those nervous centers of the muscles which have been most burdened by work, we have gathered much data, proving that intense work reacts back to an isolated or separated center, to which are attracted those factors of diseases which may eventually affect this special center or organ: in fact, lead selects the most strained parts, to exercise on them its paralyzing power; spirochæta chooses the aorta in those persons who overstrain themselves; the ectopic kidney becomes, in persons who work standing, the seat of unilateral nephritis; the radiocarpic articulation, in persons who have an occupation limiting their exercise within certain fixed motions, becomes the center of arthritis, and the radial and ulnar arteries harden in persons making great efforts with the respective joint (stone workers, filers, blacksmiths, etc.).

Under a different aspect persons working in a bended or crouching position daily, and for long hours (as brass and tin workers, and others), often complain of pains in the lumbar region which, at first, seem to be *lumbago*, but which are really due to an alteration of the spine, a real *anchylosis* induced by work done in one continual position, and by the circulatory and trophic disturbances due thereto.

In order to keep this review within limits I will only note that boys who have been engaged in hard work since childhood are frequently subject to infantilism. The Clinic has had several cases of infantilism which can be used as appropriate illustrations of the above statement.

A fund for scientific research has been provided by the contributions of the King of Italy, of his Government, of several Italian, English and American philanthropists, and of the working people of Milan.

4. CONSULTATIONS WITH THE WORKMEN

Free consultations are given every day from 10 to 12.30 to those who give evidence of being poor and hard-working people. They come:

1. To be assured as to their personal organic condition and physical fitness to learn a certain trade, to keep the one they have, or to enter into a new one.

2. To learn rules and to get medical hygienic instructions about the occupation in which they are engaged.

3. For a periodical medical inspection aiming at an early recognition of those preliminary disturbances that, directly or indirectly, arise from their occupations.

4. To have a diagnosis made and methods of treatment suggested for diseases already existing. During this free consultation service all the physicians on the Staff, each in his laboratory, are at the disposal of the consult-

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ants, so that whatever examination is called for in the different cases (blood spitting, urine, X-ray examinations, acute reactions, punctures, electro-diagnosis, serum diagnosis, etc.) can be promptly performed and all other aids such as measuring, photographs, etc., which may be useful to illustrate the case, can be provided for without delay. If the diagnosis is made at one sitting the patient is instructed in all hygienic or medical rules required by his condition; when the diagnosis is not possible at first consultation, and when the treatment cannot be followed at home, patients are received into the Clinic.

Those affected by lead colic are immediately taken in, for the following reasons:

1. In the patient's home there is lead undoubtedly, which, penetrating the system, would make the crisis longer and still more serious.

2. It is necessary to free the patient from his clothes which are usually dirty. He is then shaved and his hair and nails, where lead always collects, are cut.

3. Soon after that a very hot bath is given the patient, a treatment of pilocarpine is started by hypodermic injections, which through perspiration and salivation eliminates the lead. In this way the crisis is shortened and the pressure on the kidneys, which ordinarily in cases of saturnine colic go through a light phase of acute nephritis, is avoided.

It is the experience of the Clinic that many workmen, in whom lead poisoning is taking effect, present three orders of disturbances, viz.: paleness, dyspepsia and weakness. Our advice to them is to stop work for some days and observe a scrupulous cleanliness. After 10 days a remarkable improvement is noted, due to the elimination of lead which is not replaced. Another result of the investigations made is that persons over 50 years of age should not engage in occupations of any kind where there is a possibility of saturnism, because they become more easily affected by it, and in very serious forms (as nephritis, arteriosclerosis, paralysis, glandular insufficiency).

Children of persons affected by saturnism should never attempt to work in trades which would put them in contact with lead.

Our consultation statistics show that 8040 working people have been examined in 3 years and 11 months.

5. INSPECTIONS AND INVESTIGATIONS

The activity of our consultation service extends also to examinations of working people in their homes and to investigations concerning certain trades, on request of the manufacturers, the State, or the workmen themselves. I point out as an example worthy of praise the case of the Milanese varnishers who, every 2 weeks, undergo an examination in their meeting place, and that of the printers who also submit themselves regularly to the professional visit of the professors or assistants attached to the Clinic. A fact worth men-

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tioning occurred in connection with the strike of the lead capsule and label makers, when the Clinic demonstrated that a large percentage of the strikers were affected by saturnism, thus inducing the manufacturers to make them better propositions as to wages, etc.

During the few years of existence of the Clinic, the following researches have been conducted by Dr. Carozzi, Chief of the Investigation Department:

"Hygienic-sanitary conditions of the silk industry."
"Investigation of the polygraphic industry in Italy."
"Contribution to the so-called anæmia of the photographers."
"Hygienic protection of workmen engaged in graphic arts."
"Factory work taken home and its relation to tuberculosis."
"Infant mortality in relation with parents' occupation."

A very remarkable investigation is the one conducted by Dr. Peri on the "Professional Pathology of Dramatic Artists."

6. POPULAR PROPAGANDA ON OCCUPATIONAL HYGIENE

I am very glad, indeed, to have opened the Clinic (which was formerly intended only for physicians) to the public in general, and especially to the working class. At first we used to give general lectures on the various occupational diseases, but now we prefer to speak about each trade and profession, because in this way the attention of the workmen is more easily attracted and their interest won far more readily; so we try in every lecture to illustrate very plainly the pathology and hygiene that varnishers, decorators, printers, typists, glove makers, nurses, type founders, etc., should know, and for this we draw upon the experience and material accumulated by the Clinic, which affords illustrations of actual cases of the life and labor environment of the people. It is convincing to them to show what a powerful foe to the health and hygiene of workers alcohol is, by exhibiting at our popular lectures the bowels and other internal organs of hard-drinking workmen who, before dying, donated their bodies to the anatomic collection of the Clinic.

The *Museum of Labor Hygiene and Pathology* possesses many specimens (as shown in the hygienic exhibition of Dresden), tables, drawings, anatomic pieces and photographs illustrating unwholesome labor environments, pathological cases, etc., and these have been drawn upon to great advantage in our popular propaganda. This Museum, which is new, is open to the public on holidays from 10 to 12 A.M., and I hope that in the future it will have sections for general phenomena as *strain, fatigue, alimentation, alcohol, dusts,* and others dedicated to the various categories of the trades of Milan and its suburbs, so that proprietors, manufacturers, and employees may find in each single section all that physiology, pathology, hygiene, statistics and social assistance have to offer in their respective trades, occupations and industries. I believe that, by acting chiefly upon employers and workmen,

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we can more easily attain the purpose of eliminating from the trades all that is dangerous to the body and sometimes to the spirit of the laborer.

The expenses of the Clinic (teaching, laboratories, out-patients and assistance to 45 patients) requires a yearly output of 100,000 lire; the annual balance shows a deficit of 20,000 lire, which is covered by contributions.

Summing it all up in a sentence, the Clinic of Milan, by its work and propaganda, is a daily illustration by work and deed of the wisdom contained in Ramazzini's motto: "*Medici munus plebeios curantis est interrogare quas artes exerceant;*" teaching to physicians the technic of specific exploration, of diagnosis and of prevention, as well as that of treatment, it aims at educating them to detect accurately in the infirmities of working people what today is to be traced back to trades, and to avoid in the future—if not totally, at least in a great proportion—the evil consequences of such unwholesome trades; and it works for the extension of education and of class collaboration, availing itself of the coöperation of willing workmen and of all who are prompted, by noble feelings, to take an interest in the hygiene of work.

In fact the workmen of Milan love the Clinic and from time to time give it financial support, showing how they trust and respect it, because, being under no political influence, it has only one rule: the attainment of whatever truth mortals can reach, and only one method: to observe and to experiment.

Will other Clinics like this be established? I hope so, yet I cannot but see the difficulties that may arise. Milan enjoyed especially favorable conditions when the Clinic was decided upon; the historical period 1901–1902 was particularly appropriate to give it impetus, and the lack of a Faculty of medicine in the city greatly facilitated the task because, generally, Faculties either are too busy with the branches of teaching already existing (which are themselves not well equipped) or are misoneistic; moreover, the opening of a *Labor Clinic* does not, at first sight, gain favor, as it looks rather like a revolutionary institution, when compared with the old academic grouping of the clinical world. It concerns itself with the organs of the altered functions rather than with the causing agent. As there already exist courses of teaching named after the cause of disease (syphilology, bacteriology, clinical parasitology) or after the regions of origin of some sicknesses (tropical diseases), and that in other fields, as for instance in commerce, there are already schools of every kind, should labor, which is one of the greatest factors of life and which in fact ought to be studied from all its angles so as to be made a wholesome cause of happiness, remain without its own medical hygienic institutions?

But, I repeat, a Clinic for the pathology of work cannot be a mere infirmary for persons affected by saturnism or mercurialism and other common forms of occupational diseases; it must be a vast and complex institution, able to cope with the majority of biological, clinical and hygienic problems arising from work, and aim to be able to provide for ample means of study of good clinical material, by a complete staff. I hope that in due time this

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Clinic of Milan, the first established, may become, little by little, the social medico-hygienic labor faculty, a mixed international school open to every one, physician or not, to ill or healthy persons, to all who are willing to contribute toward the realization of healthier and more physiological occupations, which is the dream of all who are kindly hearted.

On the corner-stone of the Clinic of Milan this is carved: *in aliis vivimus, movemur et sumus.*

STATISTICS

The patients received in the Clinic number to 1100, affected as follows:

Saturnism (of which 46 are affected by chronic nephritis)	343
Other professional intoxications	20
Tubercular affections of the bronchia and of the lungs	112
Tuberculosis	23
Tuberculosis with pneumokoniosis	12
Miners' phthisis	8
Pellagra	81
Heart diseases	80
Aortitis, arteritis, and phlebitis	47
Aneurism	6
Renal ectopy and other affections of the urinary organs	88
Blood and miscellaneous diseases	144
Joint and muscle affections	21
Nervous system disturbances	67
Basedow's disease and other similar forms	12
Ankylostomiasis	12
Caissonism	7
Bone affections	7
Skin affections	7
Stomach, intestines, liver, spleen, spine affections (spondylosis)	3

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DIVISION II

Statistical Studies Relating to Occupational Diseases

CHAPTER I

MORTALITY FROM PULMONARY TUBERCULOSIS IN DUSTY OCCUPATIONS

BY FREDERICK L. HOFFMAN, LL. D., Newark, N. J.

Of the 44,130,000 American wage earners of both sexes, as estimated for the year 1915, probably at least 5,600,000, or 12.7 per cent., work under conditions more or less detrimental to health and life on account of atmospheric pollution, or the relatively excessive presence of atmospheric impurities predisposing to, or accelerating, the relative frequency of tubercular and respiratory diseases. The vast army of men and women exposed to the risk of ill-health in industry on account of atmospheric impurities caused by dust, fumes, or gas approximately divides itself as follows:

NUMBER OF PERSONS ENGAGED IN OCCUPATIONS MORE OR LESS EXPOSING TO INJURIOUS DUSTS, GASES OR FUMES, IN THE UNITED STATES, 1915 (AGES 10 YEARS AND OVER)*

Exposure to	Males	Females	Persons
Metallic dust	847,689	45,387	893,076
Mineral dust	756,459	16,424	772,883
Vegetable fiber dust	152,999	22,467	175,466
Animal and mixed fiber dust	638,997	494,505	1,133,502
General organic dust	500,936	214,235	715,171
Municipal dust	702,251	180	702,431
Gas exposure, fumes, etc	1,196,191	19,954	1,216,145
Total	4,795,522	813,152	5,608,674

The present discussion is limited to the so-called dusty trades. To a not inconsiderable extent, however, other atmospheric impurities, such as gases and fumes, are also injurious to heatlh and longevity, but the effects on the organism are not such as to result in a material increase in disease liability to tubercular and respiratory diseases. Dust is a convenient term, which includes a multitude of widely varying sources of visible and determinable atmospheric impurities. Dust is anywhere and everywhere, but the proportion of injurious particles in the air, as ascertained, for illustration, in city or municipal dust, or in the air of schoolhouses, places of worship, etc., is relatively low, due, of course, to the rapid dispersion of the minute particles in an

* Estimate based upon the U. S. census of 1910 and the rate of increase, 1900–1910, of all occupied males and all occupied females.

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area of unlimited extent. Streets and roads frequently suffer serious dust pollution, and much more at present on account of the introduction of mechanically propelled vehicles, which are rapidly taking the place of vehicles drawn by animals. Even in the best kept homes a certain amount of atmospheric dust pollution is inevitable, and in the absence of effective methods of ventilation the relative amount of impurities may attain to serious proportions; especially is this true of libraries, where dust accumulates on bookshelves and where the books themselves may be carriers of pathogenic bacteria. It is necessary to keep these facts in mind in considering the larger problem of industrial dust, since the health-injurious consequences are not entirely traceable, or attributable to the latter, but in a measurable number of cases the real harm to the respiratory organs may have resulted from atmospheric impurities out of doors, in the home, or in public places of entertainment, worship, and instruction.

I. Definitions of Industrial Dusts

It is in industries that the dust problem assumes the greatest practical importance, and three distinct classes of dust have been described and precisely differentiated by Charles Baskerville, Ph. D., in a dissertation on "Air Impurities: Dusts, Fumes, and Gases," in the New York Medical Journal for November 23 and 30, 1912.

First, as regards *insoluble inorganic dusts*, it is said that "this class includes metals (antimony, arsenic, type metal, brass, bronze, copper, aluminum, iron, steel, lead, manganese, vanadium and ferrovanadium, silver, tin, zinc, and solder) in a state of fine division (dusts, atomized metals, metallic powders); flue dusts; various ore dusts (iron ore, etc.); silica, sand, emery, flint, glass powders; carbon graphite, diamond, coal, soot; brick dust, marble, granite, cement, terra cotta; lime, gypsum, plaster, meerschaum; phosphates, guano, etc." The continuous and considerable exposure to the inhalation of insoluble inorganic dusts, according to Baskerville (who is sustained by numerous other authorities) may result in fibrosis of the lungs, chiefly because of the inhalation of siliceous or metallic particles, as, for example, is the case in the so-called potter's asthma and grinder's phthisis. Pneumonia has been reported as frequent among workmen in blast furnaces, owing in part, directly or indirectly, to the inhalation of slag dust. The disease known as siderosis is commonly met with among metal polishers, knife grinders, and others engaged in metal working.

Second, *Soluble Inorganic Dusts*.—This class, according to Baskerville, includes such substances as are likely to be swallowed and absorbed, as, for illustration, metal particles, including lead, brass, copper, zinc, arsenic, mercury, and silver, as well as soluble inorganic salts. Many dusts of this class, it is pointed out, "are dangerous not only because of their irritating or poisonous properties, but also because of their inflammability, *e.g.*, potassium chlorate."

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Third, Organic Dusts.—This class is defined as comprising such widely varying materials as "sawdust, fur, skins, feathers, broom and straw, grains and flours, jute, flax, hemp, cotton, wool, carpet dust, street sweepings, tobacco-box dust, hides and leather, felts, rags, paper, horsehair, etc." Typical of the diseases caused by organic dusts are: flax dressers' disease, a kind of pneumonia due to the inhalation of particles of flax; alkaloidal poisoning from African boxwood by workmen engaged in shuttle making; and malignant pustule and a febrile disease among rag sorters."

II. Air Analysis

This formidable array of health-injurious conditions arising out of dust which, by its mechanical properties, results (1) in a chronic irritation of the respiratory and pulmonary organs and (2) by the lesions caused, predisposes to tuberculosis, frequently with disastrous results, suggests the exceptional practical importance of extended and thoroughly qualified consideration of every aspect of the question under discussion.

The amount of mischief done, however, is not limited to the mechanical and chemical properties of industrial dust, but additional thereto the microbic contents of the air require to be taken into account. This aspect of the industrial disease problem has been considered with admirable thoroughness by Winslow and Browne, in behalf of the New York State Commission on Ventilation. These authors, in a contribution to the Monthly Weather Review for July, 1914, provide a table showing the percentages of microbes per cubic foot of air in samples from various sources, as shown in the table below:

MICROBES IN AIR FROM VARIOUS SOURCES
(Gelatin, 20°C.)
(Percentage of samples in each case)

Number of microbes per cubic foot	Country	Offices	City	Schools	Factories
0–25	44	42	28	24	12
26–50	25	23	24	17	15
51–75	11	9	16	14	20
76–100	7	2	6	13	6
101–125	7	4	8	10	29
126–150	3	2	4	7	3
151–175	....	2	3	4	....
176–200	....	4	1	2	6
201–225	1	1	2	2	....
226–250	2	1	2	2	....
251–275	....	....	3	1	....
276–300	....	1	....	1	3
301–325	....	....	....	1	3
326–350	....	1	....	....	3
351–375	....	1	1	....	....
376–400	1	....	....	....	....
Over 400	....	4	2	2	3

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The extremely interesting and suggestive fact is disclosed by this table that the country air is evidently much freer from microbic life than that of the city streets, and that the factory air has clearly the largest amount of microbic contents as determined by this method.

Air analysis is a difficult and always highly technical procedure. As yet few qualified investigations have been made to determine with approximate accuracy the actual atmospheric conditions in a large number of industrial plants under varying weather conditions and as modified by widely divergent methods of ventilation and dust control. It has properly been pointed out at the outset that ordinary air itself is more or less polluted, but the outside atmosphere is practically free from the common impurities of the air in industrial establishments, surcharged with minute particles of inorganic or organic matter which, under given conditions, may, in fact (as, for illustration, in cement or wheat flour), constitute the object of the industry itself. In this connection the following very interesting observations on air pollution may be quoted from "A Study of the Hygienic Condition of the Air in Textile Mills with Reference to the Influence of Artificial Humidification," in the Monthly Bulletin of the Massachusetts Board of Health for July, 1913:

> "Ordinary air always contains a greater or less amount of both dead and living matters in suspension which may have an important influence upon the health. The dead matter or dust, consisting of particles of mineral matter from the streets and pavements and from vehicles and machinery, and of dead organic matter from the floors and walls, from clothing and from the emanations of men and animals, especially when present in excessive amounts, irritates the mucous membrane of the lungs and respiratory passages and renders them more susceptible to invasion by the germs of disease. The living matter consists of bacteria, yeasts and moulds which are always present in greater or less numbers, and which have found their way into the air attached to dust particles, or have been projected directly into the air from the body by the acts of coughing and sneezing. So far as is known the yeasts and moulds have little pathological significance, but it is well recognized that the germs of tuberculosis, pneumonia, influenza and perhaps other diseases are frequently transmitted through the air."

The general conditions of atmospheric pollution simply tend to exaggerate or increase the observed injurious consequences of continuous and considerable exposure to industrial dust.

III. Occupation Mortality Statistics

The aggregate mortality from tuberculosis in the continental United States for the year 1915 may be conservatively estimated at 150,000, of which about 130,000 are deaths from pulmonary tuberculosis, and about 20,000, from other forms of tuberculosis, chiefly acute miliary tuberculosis, tuberculous meningitis, and abdominal tuberculosis. The tuberculosis death rate of the registration area for the year 1913 was 147.6 per 100,000 of population, which contrasts with a rate of 201.9 for the year 1900. The decline in tuberculosis is one of the most gratifying evidences of modern sanitary and public

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health progress, and while attributable to a large variety of important causes and altered social and economic conditions, it is probably safe to assume that a not inconsiderable share in this reduction of the death rate is to be assigned to the very material improvement in the conditions or methods of factory life and the higher regard paid to the physical health and comfort of the employees. As a general introduction to the scientific study of the problem of tuberculosis in industry, it is essential to take note of the wide disparity in the relative death rates by age and sex. The table following shows the rates as determined for the United States registration area for the period 1909–13. The rates are limited to pulmonary tuberculosis, and include an aggregate of nearly 400,000 deaths of both sexes during the five years under observation:

Comparative Mortality from Pulmonary Tuberculosis
U. S. Registration Area, 1909–1913
(Rate per 100,000 of population)

Ages	Males	Females
Under 5 years	33.7	29.7
5–14 years	10.8	20.6
15–44 years	184.6	165.5
45–64 years	227.0	120.1
65 and over	195.3	148.9
All ages	145.2	116.2

This table is of exceptional interest and importance. It is shown that at ages under 5 years the rates are about the same for both sexes, but at ages 5–14 the female rate is nearly twice the male rate. During the age period 15–44 the male rate but slightly exceeds the female rate, but nearly twice the relative mortality from pulmonary tuberculosis occurs among men at ages 45–64, when compared with the corresponding death rate for women. At ages 65 and over the rates approach each other, but there remains a considerable excess in the relative mortality of males. Combining all ages, the excess in the male rate is 29.0 per 100,000 of population, but the excess at ages 45–64 is 106.9, or equivalent to 1 per 1000. There is no escape from the conclusion that under the known conditions of industrial life, and the relative age distribution of wage earners of both sexes, *this excess in the mortality from tuberculosis among men of ages 45–64 is largely, if not exclusively, the result of health-injurious conditions in industry,* enhanced, no doubt, more or less by habits and a more general indifference to the requirements of personal hygiene.

The differences in the mortality from pulmonary tuberculosis by no means measure the full extent of the higher mortality among adult males, due more or less to the strain and stress of industrial life. The mortality from respiratory diseases is also higher among men, and especially from lobar pneumonia.

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For all respiratory diseases combined the male death rate in the registration area during 1908–12 was 185.0 per 100,000, in comparison with a rate of 163.7 among women. Since it is frequently asserted that, in a large measure, the higher death rate of males from pulmonary tuberculosis in adult life is the result of health-injurious habits, rather than health-injurious occupations or industrial conditions, it is significant that the differences in the mortality from lobar pneumonia, which is a much better indication, should be so much less than in pulmonary tuberculosis.

IV. Relative Incidence of Tuberculosis in Dusty Trades

Proceeding from these general considerations to the special problem of the mortality from tuberculosis in dusty trades, it has seemed best to present the results in a series of tables, which are the underlying data of a series of charts furnished to the U. S. Bureau of Labor Statistics as part of a collective exhibit prepared for the Panama-Pacific International Exposition. The tables are derived exclusively from the industrial mortality experience of the Prudential, and while limited to the proportionate method, they conform, in the main, to similar results arrived at by means of rates calculated in accordance with more precise and satisfactory methods of statistical inquiry. It, unfortunately, is not possible to correlate the deaths by occupations in the Company's industrial experience to the exposed to risk. For the purposes of industrial hygiene, however, the proportionate method is quite satisfactory, and perhaps even more so when the fact is considered that by this means the exact quantitative amount of mortality from a specified group of diseases is shown in its relation to groups of occupations or particular employments. On the basis of the Company's experience for 1907–1912, including 162,765 deaths of occupied males, the proportion of deaths from tuberculosis of the lungs at ages 15–24 was 34.2 per cent. of the mortality from all causes in this group; at ages 25–44 it was 37.4 per cent.; at ages 45–64 it was 13.5 per cent.; and at ages 65 and over it was 3.1 per cent. Relatively as well as actually, therefore, tuberculosis of the lungs was of most importance at ages 25–44, which, broadly speaking, coincides with the period of greatest productive efficiency on the part of wage earners collectively considered.

The experience has been arranged according to the character of the dust, in six groups, as follows:

1. Metallic dust, including 3,374 deaths from all causes, and 1,208 deaths from tuberculosis.

3. Mineral dust, including 3,734 deaths from all causes, and 1,037 deaths from tuberculosis.

3. Vegetable fiber dust, including 1,120 deaths from all causes, and 344 deaths from tuberculosis.

4. Animal and mixed fiber dust, including 1,276 deaths from all causes, and 372 deaths from tuberculosis.

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5. General organic dust, including 5,694 deaths from all causes, and 1,140 deaths from tuberculosis.

6. Municipal dust, including 10,567 deaths from all causes, and, 3,021 deaths from tuberculosis.

In the group considered chiefly with reference to exposure to *metallic dust*, 16 specific occupations are given. At ages 15–24 the proportionate mortality from *tuberculosis of the lungs* was 45.2 per cent.; at ages 25–44 it was 49.7 per cent.; at ages 45–64 it was 21.1 per cent.; and at ages 65 and over it was 5.4 per cent. The corresponding proportionate mortality from *respiratory diseases* in this group at ages 15–24 was 7.0 per cent.; at ages 25–44 it was 8.9 per cent.; at ages 45–64 it was 13.6 per cent.; and at ages 65 and over it was 14.8 per cent.

In the group considered chiefly with reference to exposure to *mineral dust*, 13 specific occupations are given. At ages 15–24 the proportionate mortality from *tuberculosis of the lungs* was 33.1 per cent.; at ages 25–44 it was 41.1 per cent.; at ages 45–64 it was 22.7 per cent.; and at ages 65 and over it was 7.0 per cent. The corresponding proportionate mortality from *respiratory diseases* in this group at ages 15–24 was 13.6 per cent.; at ages 25–44 it was 11.5 per cent.; at ages 45–64 it was 14.9 per cent.; and at ages 65 and over it was 16.1 per cent.

In the group considered chiefly with reference to exposure to *vegetable fiber dust*, 12 specific occupations are given. At ages 15–24 the proportionate mortality from *tuberculosis of the lungs* was 41.2 per cent.; at ages 25–44 it was 49.5 per cent.; at ages 45–64 it was 21.3 per cent.; and at ages 65 and over it was 4.6 per cent. The corresponding proportionate mortality from *respiratory diseases* in this group at ages 15–24 was 10.8 per cent.; at ages 25–44 it was 6.3 per cent.; at ages 45–64 it was 13.7 per cent.; and at ages 65 and over it was 13.9 per cent.

In the group considered chiefly with reference to exposure to *animal and mixed fiber dust*, 8 specific occupations are given. At ages 15–24 the proportionate mortality from *tuberculosis of the lungs* was 46.3 per cent.; at ages 25–44 it was 49.7 per cent.; at ages 45–64 it was 17.5 per cent.; and at ages 65 and over it was 3.1 per cent. The corresponding proportionate mortality from *respiratory diseases* in this group at ages 15–24 was 9.9 per cent.; at ages 25–44 it was 10.1 per cent.; at ages 45–64 it was 11.6 per cent.; and at ages 65 and over it was 12.9 per cent.

In the group considered chiefly with reference to exposure to *general organic dust*, 15 specific occupations are given. At ages 15–24 the proportionate mortality from *tuberculosis of the lungs* was 38.6 per cent.; at ages 25–44 it was 43.1 per cent.; at ages 45–64 it was 14.6 per cent.; and at ages 65 and over it was 3.5 per cent. The corresponding proportionate mortality from *respiratory diseases* in this group at ages 15–24 was 7.6 per cent.; at ages 25–44 it was 10.7 per cent.; at ages 45–64 it was 12.8 per cent.; and at ages 65 and over it was 14.1 per cent.

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In the group considered chiefly with reference to exposure to *municipal dust*, six specific occupations are given. At ages 15–24 the proportionate mortality from *tuberculosis of the lungs* was 36.1 per cent.; at ages 25–44 it was 39.7 per cent.; at ages 45–64 it was 16.2 per cent.; and at ages 65 and over it was 3.1 per cent. The corresponding proportionate mortality from *respiratory diseases* in this group at ages 15–24 was 8.2 per cent.; at ages 25–44 it was 10.4 per cent.; at ages 45–64 it was 13.3 per cent.; and at 65 and over it was 15.3 per cent.

V. Miners' Phthisis

More intensive statistical investigations yield results of even greater practical value. As yet, however, not many industries and specific occupations have been made the subject of extended specialized statistical and medical consideration. Any single disease factor is bound to become obscured where a number of other determining circumstances modify the results. Age, sex, race, and even climate require to be taken into account. Miners' phthisis is an excellent illustration of an industrial disease common to an industry only under exceptional conditions. Modern rock drilling, particularly in siliceous rock, is an exceptionally dusty operation, followed invariably, unless subject to control, by extensive injuries to the health of persons employed underground. Among coal miners tuberculosis is very rare. Among metal miners working in quartz rock the disease is very common. In Montana, regardless of a high proportionate mortality from accidents, the percentage of deaths from tuberculosis was 10.8 at ages 15–24, 28.5 at ages 25–44, 35.0 at ages 45–64, and 9.7 at ages 65 and over. These interesting statistics are based upon an original examination of the mortality records of Butte, Mont., for 1907–1911. Conditions are even worse in the Joplin zinc-and-lead mining district of Missouri. As the result of official investigations it has been shown that the prevalence of tuberculosis and fibrosis in southwestern Missouri is closely related to excessive dust in sheet-ground mines. As shown by a report made by A. J. Lanza, M. D., of the U. S. Public Health Service, and Mr. Edwin Higgins, mining engineer of the U. S. Bureau of Mines, the dust is caused by various practices but chiefly by the blowing of dry holes. The recommendations offered as the result of this investigation are of such exceptional practical importance that they may safely be considered as a first requirement for the effective sanitary control of health-injurious conditions in the practice of metal mining in general.

1. Means should be employed for the abatement of rock dust in the mines as follows: Provide a water supply for every working face, preferably by the laying of separate water lines; as rapidly as may be, without too great cost, equip all miners with some type of drilling machine which provides for water passing through the core of the drill into the drill hole, although results almost as good may be obtained by equipping the present type of dry drill with a water spray; make and strictly enforce rules against squibbing and

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boulder popping during the time that the shift is underground, and against the blowing of dry holes at any and all times; thoroughly wet the working faces and the broken rock every morning and again at noon if necessary; improve ventilation by the sinking of new shafts whenever practicable.

2. Do away with common drinking cups and kegs, and water pipes which allow the miner to bring his lips in contact with the orifice.

3. Do not employ as shovelers men under 20 years of age.

4. Through coöperation among the operators provide a maximum daily tonnage for shovelers so that they cannot injure their health through overwork.

5. Provide a warm, dry and clean place in which the miners may change their clothes.

6. Through intensive educational campaigns in the public schools and among the miners themselves disseminate information as to the harmful effects of insanitary practices and conditions, such as crowded living quarters, overwork, exposure, dissipation, the breathing of air polluted by powder fumes and rock dust, the use of common drinking devices, etc.

The most scientific and exhaustive investigations into the problem of miners' phthisis and pulmonary tuberculosis have been made in South Africa. In a recently revised report of a commission appointed under the provisions of the "Miners' Phthisis Allowances Act," including a statistical appendix by Dr. G. G. Maynard, all the essential facts of the problem are clearly presented with reference to conditions not only in South Africa but elsewhere throughout the world wherever deep mining in quartz rock is carried on to a considerable extent. (For a copy of the act, "To make provision for persons who have contracted miners' phthisis," by the Union of South Africa, see Bulletin of the International Labor Office, Vol. IX, Nos. 1–2, 1914.)

VI. Industrial Phthisis

The more special aspects of the industrial dust problem cannot be advantageously discussed with brevity. Every dusty trade presents a problem in itself. As a first prerequisite for an improvement in existing health conditions in industry it is essential that the exact conditions and processes be thoroughly understood and made a matter of record. As a typical illustration of what is necessary in this direction a brief reference may be made to the "Survey of Industrial Health Hazards and Occupational Diseases in Ohio." The first results of this investigation, made under the direction of E. R. Hayhurst, M. D., and published by the Ohio State Board of Health, constitute a treatise on the subject of industrial diseases. As pointed out in this report, "Dust may be inhaled or ingested, or affect the skin, the eyes and the ear canals. The daily subjection to dust, for more than brief intervals at a time, is always damaging. The skin and the eyes may become physiologically inured to it, but not so with the internal organs. The least

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harmful dusts are those arising from the natural earth itself, such as the farmer is subjected to, although there are many exceptions to this in the case of alkali, sandy, or stony earths, etc. White flour and starch appear to be practically harmless to the normal person, soapstone dust and talc may be placed next in order, but a tuberculously inclined person subject to these, if they do no more than irritate the nose and throat and promote coughing, is almost certain to see an increment in his disease. Next in order of harmfulness come wood dust, bran dust, coal dust, clay dust, ore dust, mineral dust and stone dust. It will be seen that the organic dusts are the least harmful. Dusts in general produce a chronic catarrh of the respiratory and digestive organs. This leads to a fibrosis, which is the same process that is gradually brought about by old age. These catarrhs and fibroses result in lowered resistance of the damaged parts and invite secondary diseases, which are usually the cause of death."

In continuation of these interesting observations, which are amplified by illustrations of exhaust systems for dust removal in polishing departments, iron and steel works, etc., it is stated:

"Two-thirds of a pint of coal dust has been found in the lungs of a former coal miner. One-third of the weight of the lungs of a rock-driller has been found to consist of rock dust. Probably the most harmful dust of all, with the exception of poisonous dust, is emery dust, which is composed of exceedingly hard, crystalline, sharp particles; next to this comes sand or sandstone dust, to which workers are subjected in surfacing, polishing and crushing stone, in sand-blasting, etc. No person should work in a dusty atmosphere.* Either by mechanical means, wet processes, and modification of processes, or by personal hygiene, as respirators, etc., dust can be kept out of the human system. Dry sweeping during work hours is a most vicious practice. Vacuum cleaning not only for floors, but in many dusty processes, is especially recommended. In dust-mixing processes much of the material can be handled by vacuum pipes instead of scoop shovels."

VII. General Conclusions

There is at the present time a most regrettable neglect on the part of practically all local health authorities to take proper cognizance of the health-injurious consequences of dust and to insist upon measures of effective prevention and control. The initial work which has been done in this respect by the State Board of Health of Massachusetts and the State Department of Labor of New Jersey is deserving of most careful consideration. As yet, however, no state or local board of health, or department of labor and factory inspection, in the United States has followed the thorough-going English methods, as best emphasized in the annual reports of the Chief Inspector of Factories. The work of this department is intelligently coördinated to the efforts of local boards of health, as perhaps best illustrated by the ex-

* This suggestion of course must not be literally construed, since all atmosphere contains more or less dust, some of which is naturally relatively harmless. By a dusty atmosphere is meant one which is contaminated by dust of known or ascertainable health-injurious qualities.

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tended consideration of the tuberculosis problem in Aberdeen, with special reference to occupation, and particularly the stone industry, which is of considerable extent in or near to the city of Aberdeen. Mention also requires to be made of what has been done by the United States Bureau of Labor Statistics in giving publicity to the results of special investigations into the mortality from tuberculosis in dusty trades, and to the American Museum of Safety for exhibiting dust-preventive devices and models of industrial diseases, including fibroid phthisis. A concise summary of the entire subject of dust and fumes as foes of industrial life is contained in an address delivered before the 15th International Congress on Hygiene and Demography by Sir Thomas Oliver, M. D., of Newcastle-upon-Tyne.

The statistical aspects of the problem have been discussed briefly in my address on "Industrial Accidents and Trade Diseases in the United States," delivered on the same occasion, and earlier in my two papers on the mortality from consumption in dusty trades, published respectively as Bulletins 79 and 82 of the U. S. Bureau of Labor. The results of all inquiries into the problem of industrial dust emphatically and unequivocally suggest the supreme importance of a clear realization on the part of employers and employees on the one hand and state or local health authorities on the other, that a vast amount of irreparable damage results from indifference and neglect in the control of the dust nuisance, the harmfulness of which is but imperfectly realized on the part of those most seriously affected thereby. The solution of the problem will depend largely upon the intelligent and hearty coöperation of federal, state, and local health authorities, the labor bureaus or industrial boards, and most of all the employers and employees, who are most familiar with the conditions under which modern industry is carried on. The effective control of the dust factor in industry and the gradual elimination of health-injurious dusts from certain well-defined industrial processes must, in course of time, profoundly affect the tuberculosis death rate and result in a material reduction of the mortality, to the inestimable advantage of the community, the industries and, most of all, the workmen themselves.

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CHAPTER II

THE USE AND THE FALLACIES OF STATISTICS

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The Use and the Fallacies of Statistics.—Statistics are facts expressed in numbers, collected and arranged for study. When there are few facts to be considered statistics are not necessary; the mind does not need this aid. Suppose one asks the wages of the five men who work for Smith. The reply is easily given and understood if the facts are known: Tom gets $15 a week, George and Henry receive $12 each, and Bill and Mike get $10 each. But suppose one asks the wages of the laborers in a large factory where several thousand men are employed. We might go through the list in the same way—in fact the paymaster is obliged to do so—but for purposes of study we must generalize and say that so many men get $15 a week each, so many more get $12 each, and so on, expressing the results in figures. This is the statistical method—to enumerate, to classify and to compare.

As the number of different facts to be considered in any problem increases, the need of studying them by the numerical method becomes constantly greater and we are obliged to resort to arithmetical processes of various kinds. Sometimes we are compelled to use quite complicated mathematical methods, but the mathematics required in most statistical work are very simple.

The great danger in all this work is that in our consideration of the numbers we may lose sight of the facts for which they stand, in the same way that the countryman could not see the city because there were so many houses. Failure to visualize is the besetting sin of those who deal with facts in this abstract way. But if it is hard for the statistician it is even harder for persons who are not familiar with statistical methods to visualize when the facts enumerated are scattered in time or place. If a factory burns and a dozen girls are killed it is regarded as a tragedy, people shudder and demand safeguards that will make a repetition of the accident impossible; but if the same number of girls had been killed one at a time in different factories from the same lack of adequate fire protection the world would make no comment, because it is the usual order of nature for people to die one at a time. It is one of the tasks of the statistician to bring together such scattered facts so that they may be visualized and their true significance made known. Thus it happens that occasional typhoid fever epidemics tend to reduce the total number of deaths from this disease; that occasional railroad accidents tend to make travel safer; and that occasional conflagrations tend to reduce the total loss of life and property from fire. In other words, because of our

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failure to visualize the scattered facts our actions are controlled more by the occasional occurrence of a striking incident than by the prosaic little happenings which in the aggregate are of much greater importance. Constant dropping wears away the stone. It is part of the vital statistician's work to enumerate the drops, that the magnitude of the force at work may be appreciated. It is his function to induce people to substitute action based on judgment for action based on emotion.

The statistical method is always found to be popular and useful when people are interested in the facts considered. The boys of the country are statistical encyclopedias when baseball and batting averages are considered; merchants follow the statistics of prices and sales with an eye keen to the main issue; while the politician knows almost by heart the votes for all the candidates in the election districts under his charge. Yet, taken as a whole, it cannot be said that the art of using statistics has become so well developed that the general public appreciates it. In fact, the contrary appears to be the case. President Kirkland of Harvard College used to say: "the chief use of statistics is to refute other statistics," and everyone has heard the apothegm that "figures will not lie, but liars will figure." There are several reasons for this state of mind in regard to the use of statistics, but three of them stand out prominently: first, the data are not presented clearly and in an interesting way; second, the data are not used in a logical manner; and third, the reader fails to make the necessary mental effort to understand and visualize the facts for which the numbers stand. The writer believes that vital statistics, in order to be of practical benefit, must be used with truth, with imagination and with power.

Again, we may apply to statistical proof the two fundamental requisites which we learned in our study of geometry and which are expressed in the words "necessary and sufficient."

The Statistical Method.—The statistical method involves three fundamental processes—enumeration, classification and comparison. Or, as the logician might say, we collect the data and then abstract them. In making these studies we resort to various devices; we arrange our data in tables; we generalize from these tabulations by computing averages and medians and modes; we illustrate our facts by diagrams and cartoons; we compare the results of one class or group with those of another class or group; we express these relations by mathematical formulæ and thus determine certain laws or show the absence of a law. In all of these processes we must be ever on our guard against fallacies that lurk therein. Let us consider some of these.

Enumerations.—Enumeration is counting—an easy process, one says. So it is if we know what is to be counted. Here, at the outset, we meet our first difficulty. What are we going to count? It depends upon the definition of the unit under consideration. Suppose we are enumerating the dwelling-houses in a city. What shall we regard as a dwelling house? Is a church a dwelling-house if the sexton lives in it, or a stable if the hostler

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dwells there? Is a single building with two front doors one dwelling-house or two? Is a "three decker" one house or three? It will be seen that the term "dwelling-house," familiar as it is to us, needs exact definition if it is to be used as a basis for statistical study.

Again let us suppose that we are studying the effect of the environment in a cotton mill on the health of the operatives. Whom shall we consider to be an operative? Shall we include only those who work in the rooms where cotton is made or shall we include also the machinists and packers and the watchmen and the clerks in the office? And if we take only the cotton workers shall we take into account the period of time that they have been employed? If a man has worked in the mill only a week ought he to be included? Obviously these matters must be decided before the enumeration is begun. Many statistical studies are worthless because of failure to properly define the units in the beginning. If the reader wished to obtain an illustration of the difficulty of enumerating people in well-defined groups let him ask three or four of his friends to count the number of well-dressed men or the number of beautiful women in an assembly. The results will tend to make him conservative, and perhaps pessimistic, as to the usefulness of the statistical method.

A second source of error in enumeration is failure to find the units to be counted. In taking a census some persons are never found by the enumerators. At the last census in England, where the census is taken on a given night, it is said that some of the suffragettes walked the streets all night so as not to be counted, arguing that if they could not vote they would not be counted. It is well known that birth records are incomplete in all countries, and that cases of reportable contagious diseases are not reported in full or anywhere near it. On the other hand, there is the contrary danger that the same unit may be counted twice. This may result from accident or carelessness; but padded census records, counts of tissue ballots and all sorts of tricky methods are at times resorted to by the unscrupulous. An old darkey selling eggs used to count them thus: "one, two, three, four, six, seven, eleven, fourteen," and so on. The process of enumeration which is our fundamental statistical process should be regarded as the accurate counting of well-defined units.

There is a rather peculiar form of error, which often creeps into statistical enumerations, known as the error of round numbers. Thus in recording dates of past events some will be given on the exact day of occurrence, but where memory fails, some will be given approximately as "on the first of the month," or "on the fifth, tenth, fifteenth" and so on. This causes a preponderance of numbers recorded on these days. The same is true in regard to age. A person who is 39 or 41 years of age may state his age as 40, a round number which to his mind is near enough. The tendency to this error may be discovered by finding that an undue proportion of the collected figures are divisible by 5 or 10 or 100. Another means of discovery is by plotting the data.

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Classifications.—There are three general methods of studying statistical enumerations. First, there is the *complete statistical study*, which includes a full account of all the units within the desired area or within the specified time. This method, of course, brings the surest results and can often be employed when the study is not too extensive. Often it is impracticable. Second is the *monogramic method*, a procedure in which a detailed and exact study is made of a particular group. When the group selected for study is a well chosen type the application of this method yields valuable results, but there is always a danger in generalizing from monographic researches. The third method is the *representative method*, as the method of sampling, when a selected part is taken as representative of the whole. The value of this method is dependent upon the accuracy of the sampling process, often a difficult matter to negotiate. Sampling may be by *ramdom selection*, or by *mixture and subdivision*. In any sampling process there is always a tendency to take the obvious and the accessible, and this may cause the sample to be unrepresentative; or one may be governed in his sampling, consciously or unconsciously, by some preconceived notion as to the probable results or the desired results.

The technique of statistical induction, says Royce, consists in learning, first, how to take fair samples of the facts in question and, second, how to observe these facts accurately and adequately.

In classifying the data we arrange them in sections, classes, groups, and series for purposes of comparison. Collections of units differing from other collections by characteristics not expressed in figures are termed sections or *classes*. Thus, populations are divided into classes according to sex, nationality, conjugal condition, civil divisions. Collections of units differing from other collections by characteristics expressed in figures are called *groups*. As an example, population is divided into age groups or into groups of persons having different weights or heights. In generalizing, the average is found for each group and these are compared with each other and with the whole. Data are also arranged in *series* according to some natural sequence or some order of magnitude or chronological order.

The difference between a class and a group is well illustrated by a little joke that recently appeared in one of the papers. The question was asked, "What class of people live to be the oldest?" The answer was, "Centenarians." The reader will recognize that this witty reply was not responsive to the question, in that centenarians represent a "group" of people, not a "class."

Tabulation.—For purposes of study and display the collected data are commonly arranged in tabular form. The preparation of tables is an important part of statistical work and cannot be done too well. Essential qualities of good tabular work are clearness, compactness, and neatness. In the first place every table should have a title that describes clearly what the data are. Preferably it should be as short as possible and it is good training in

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the use of words to produce an artistic title. Each column should also have a clear and appropriate title, in which well understood or well explained abbreviations may be used if it is necessary to save space. Where the heading is complex, that is, where certain parts of the heading cover more than one column, care should be taken to have this clearly indicated by proper ruling. Where different columns are referred to in the text accompanying the table it may be found convenient to give each column a serial number, from left to right, placed in parenthesis just below or above the heading. Long unbroken columns of figures are confusing to the eye and horizontal spaces between every five lines are often left. This practice is to be commended.

Ratios—Rates.—Ratios may be expressed by common fractions or by decimals. It is seldom wise to use common fractions of which the denominator is greater than 10, unless it is some commonly used multiple of 5 or 10, such as $\frac{1}{25}$ or $\frac{1}{500}$. In statistical tabulations decimals should be used. Rates are merely ratios in which the basis of comparison is some well-recognized division of area or time or some common unit of the decimal system. Examples: gallons of water per day, persons per square mile, number of births per 1000 marriages. Compound rates are commonly used when comparison is made with two bases: thus, gallons per capita per day, number of births per 1000 marriages per annum. Most of the rates used for the comparison of vital statistics are compound rates as they involve both area and time. Sometimes these are termed compound averages.

Misuse of Averages and Rates.—Fictitious accuracy should be avoided. If 35 out of 57 balls were white the percentage of white balls would be 61.40 per cent. The smallest possible error, *i.e.*, 1, would change the percentage to 59.65 per cent. or 63.16 per cent. Clearly for figures less than 100 fractions of per cents. are illogical. In the same way death rates for populations of less than 1000 are useless beyond the third significant figure. Comparisons of averages of fictitious value are also to be avoided.

Changes of basis in the computation of rates should be kept in mind in order to avoid error of statement. Thus, in the year 1880 the receipts of a water company were $400,000; between 1880 and 1890 they decreased 10 per cent., so that between 1890 and 1900 they became $396,000 (not $400,000). The basis used should be stated in words if it is not perfectly clear from the context.

When interpreting ratios it should be carefully noted whether the numerator bears a direct relation to the denominator. In proportion as it fails to do so, any inference from it is less valuable. For example, the ratio between the number of births and the total population is less close than between the number of births and the number of married females of child-bearing age. In the same way crude death rates based on total population regardless of sex or age are less useful in studying relative hygienic conditions than when age and sex are taken into account. It is failure to take this element into account that has led people to say, "You can prove anything by statistics," and that has given unscrupulous persons opportunity to deceive the unthinking or

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the uninformed. A moral responsibility of no mean order thus rests upon the statistician.

Ratios are sometimes necessarily used in an indirect way. Thus, the average annual exports and imports are taken to represent the business condition of a country. Here a part is taken for the whole. The method is proper if in the interpretation it is recognized that it is a part. Or the typhoid fever death rate of a city is taken as an index of the sanitary quality of the public water supply. It may be indeed such an index, but it is only one.

Another danger is that common to all forms of reasoning, namely, that of using *post hoc* for *propter hoc*. This fallacy is very likely to creep in unawares in statistical work under cover of apparent accuracy and thoroughness of investigation implied by the use of columns of figures. Bailey has well said that "the phrase 'other things being equal' has covered up a multitude of sins. As a rule other things are not equal." It is even worse, perhaps, to take it for granted that other things are equal. Bailey also warns against the hidden errors that may lie under the use of the terms "it is undoubtedly true that, etc.," and "it is probably the case that, etc." Of vital importance is it, therefore, to make sure that the data collected are sufficient in kind and number for the purpose for which the statistics are intended. It saves time and labor in the end to consider carefully at the outset just what data are needed. Where, as is often the case, the statistician had no control over the collection of the data, he should make every possible attempt to ascertain the reliability of the sources of information and not attempt to draw conclusions not warranted by the conditions under which the figures were collected. The statistician cannot safely write newspaper headlines.

The Index.—Averages of different groups and even of different classes are sometimes combined to give a single ratio, more convenient for use and perhaps more accurate as a measure of certain phenomena. Thus, the prices of various standard commodities sold in any year are combined to give a single figure that is taken to indicate the state of trade during that year. This may be done by taking a weighted average of the different articles sold in any one year and comparing it with the weighted average for some particular year or series of years taken as a standard. Obviously there are various ways in which an index can be prepared.

The index has not come into use to any extent in the study of vital statistics, but it would seem logical to use it in comparing the relative hygienic conditions of different cities. This is to a certain extent accomplished when crude death rates are adjusted to take into account the composition of population, as to age, sex and nationality.

Variations.—In the treatment of objects or organisms that belong to some definite group or in the observation of natural phenomena it is commonly found that the results are much alike, although not precisely alike. Thus, the heights of most men are quite near the average of all, but a few men are above the average height and a few below it. The same is true of

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the weights of persons in any age group. It is true of the ages at which people die from different causes. And so in many of the phenomena that come under the head of vital statistics many observations will be found near the average of all, but a few will be quite different. These variations commonly follow certain mathematical laws of probability which are found to be quite useful in statistical work. The study of variations is fascinating in itself. It has already played a large part in biological investigations and little by little the idea is being extended into the engineering sciences. For example, it has been found that the frequency of rainfalls of different magnitudes can be expressed in accordance with the law of probability, and the same is true of the occurrence of floods, and similar natural phenomena.

In this brief chapter it is impossible to discuss this interesting subject of variation and frequency, but the reader is urged to pursue it in the standard text-books on Statistics and Least Squares.

Death Rates.—A figure commonly used in sanitary and hygienic studies is the death rate. By this is meant the ratio between the number of persons dying in a given period of time and place, and the number of persons in the place alive at the middle of the period, referred to some round number, as 1000 or 10,000 or 100,000 as the basis of camparison. Unless otherwise specified the period of time is taken as 1 year. When considering deaths from all causes the basis of population is taken as 1000, but when considering deaths from particular causes 100,000 makes a better basis for comparison.

In a certain city in the year 1910 there were 5710 deaths from all causes. The population was 390,000, consequently for every thousand population the number of deaths was 5710 divided by 390 thousands, or 14.6. This represents the general death rate, or the crude death rate, as it is often called. Such a rate enables us to compare on the same basis the mortality for places of different size. It is a convenient yard stick, but it must be used with great caution. It is often misused. The expression "crude death rate" is to be taken literally for such a rate is indeed a very crude one.

Let us consider, for a moment, how it is made up. In the first instance it is a common fraction, and has a numerator and a denominator. The numerator is the number of deaths at all ages, from all causes, from all sorts of occupations, in all kinds of houses, the only boundary of the group being that the deaths occurred within the given place and within the period of a calendar year. Presumably, it includes all the deaths there were, but even this is not always the case. Suppose a child is born dead, or, as we say, is still-born; is this still-birth included among the deaths? In some places it has been so included. Modern statistical practice does not include it. Again, suppose a person whose home is in the given place dies away from home and is brought home for burial, or that another dies in a hospital within the given place and is buried outside of the place. Here are other chances for uncertainty. Hence, the numerator of our death-rate fraction is more or less unreliable and ambiguous.

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The denominator is equally so. Except in years when a census is taken the mid-year population must be estimated, and this estimate may be wide of the mark. The natural tendency is for population to increase at a geometrical rate, that is, to increase like compound interest, but actually the population of a given place changes from various other causes which mask this normal rate of increase, from emigration and immigration and from changes in the birth rate. These changes are often sudden and large. The United States Census Bureau makes a practice of estimating that between the census years the population in any given place increases or decreases by a constant annual increment. On the whole, this may be true and it is probably the best general rule to follow in this country at the present time, but in any given place it may be wide of the mark, and the denominator of our fraction may therefore be wrong in any particular year. But even if correct, the denominator stands for a composite population and includes the young and the old, male and female, the rich and the poor, the well and the sick, the native and the foreign. The only boundary of the group is that the persons included must be alive and living in the given place at mid-year.

The general death rate, therefore, is an imperfect measure of hygienic and sanitary conditions except between groups of population similar in character. The crude death rate of a manufacturing city like Lawrence, Mass. cannot be fairly compared with that of a residential city like Newton. The general death rate of a big city, with its large middle-aged population, cannot be compared with that of a rural county deserted by the middle-aged people who have gone to the city. Chicago cannot be fairly compared with Boston on this basis, nor New York with New Orleans. It is true that this is done repeatedly, but the figures mean but little, and the conclusions reached from such comparisons are often false and mischievous.

The fallacy in the general death rate lies partly in its liability to error, but still more in the composite character of the two quantities which together give us the ratio.

General Death Rate Influenced by Infant Mortality.—It is not generally realized that about one-fifth, to use an approximate figure, of all of the deaths in a given year are of children less than a year old. One-quarter of the total deaths are almost always children less than 2 years old. Hence, whatever influences the deaths of children influences very greatly the general death rate. An abnormally hot summer may do this or an epidemic of children's diseases.

It follows also that a high birth rate results in a higher general death rate and a comparison of the curves of birth rates and general death rates shows a surprising synchronism. Birth rates and marriage rates both fluctuate with the general death rate.

During the last quarter century the general death rates, have been falling. This has been due in great measure to improving sanitary conditions but it has been due also to lowering birth rates.

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In this connection, however, it should be remembered that the birth rate influences the denominator of the death-rate fraction as well as the numerator, for most of the children born do not die at an early age. Were the population of a place not influenced by immigration a lowering birth rate would not continually reduce the general death rate, for with fewer children born there would be a smaller population upon which to figure the death rate. The problem is endlessly complicated and requires a deep mathematical mind for its full comprehension. It is sufficient to remember that as long as the birth rate continues to fall and our present immigration keeps up so long will the crude death rates continue to fall. And when some years hence the birth rates cease to fall, as they must, and when they increase, as it is to be hoped they will, this will be accompanied by an increase in the general death rate. This increase in the death rate will be normal and must not be regarded as a sign of failure on the part of sanitation. It is hardly to be expected that the improvement in sanitation will prevent this ultimate increase in the general death rates. If sanitarians will recognize this fact to-day it may save them some heart burnings in years to come.

Infant Mortality not an Index of Sanitary Environment.—Studies of the death rates of the world show that until within a very few years there has been no substantial decrease in infant mortality, and this, too, in spite of the fact that the specific death rates for children and persons up to middle life have materially decreased. The lowering of the death rates from those diseases which are caused by specific infections have been very conspicuous for a generation, and this is clearly due to improvements in sanitation resulting from our better knowledge of bacteriology. Infant mortality does not reflect so clearly our recent efforts to obtain a clean environment. Hence, the infant mortality of itself does not offer a satisfactory index of sanitary conditions.

The decrease in infant mortality which has occurred during the last 5 or 10 years has been due rather to the various activities that come under the heads of infant welfare and motherhood training. It is important to keep the distinction between this work and the work of sanitation clearly in mind. A study was made in Boston a few years ago which showed marked variations in the specific death rates of infants at different ages. The data collected were tabulated by months and by nationalities. It was found that the Italian infants had a low death rate during the first few months, that is, during those months when the children were being nursed by the mother, but that after the age of about 9 months the death rates began to increase, a fact that was apparently due to the child entering a new and unclean environment. The children of Jewish parentage maintained a relatively low death rate for a long period. It is only by studying vital statistics in a detailed way that such facts as these are brought to light. While the processes of generalization are of value the opposite processes of subdivision and classification are equally important.

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Restricted Death Rates.—Instead of computing the general death rate of a place in the manner indicated we may restrict either the numerator or the denominator of the fraction. We may limit the time to something less than a year and thus obtain monthly rates, or weekly rates, or even daily rates. We may limit the deaths to those from a particular cause, and obtain the annual death rate from tuberculosis or from typhoid fever, or from lead poisoning. Or we may limit the group or class represented by the denominator to certain ages or occupations or nationality or sex, and in various ways we may obtain rates that are useful for study and comparison. These rates may have in common scarcely anything more than that they are referred to some round number as 100, or 1000, or 100,000 as the basis of comparison, or in other words that they are expressed by the decimal system. Such rates we term "specific rates" for the reason that they include only the data for definitely specified groups or classes.

In making up these restricted or specific death rates and in comparing them it is of the greatest importance to make sure that in restricting a group by one set of boundaries we do not at the same time and without knowing it, perhaps, restrict it by other boundaries. Thus, by dividing the instructing staff of a university into classes such as assistants, instructors, assistant professors, associate professors, and full professors, we may at the same time be establishing a grouping according to age. In dividing the workmen employed by a city into policemen and laborers, we might at the same time be dividing them by nationality into Irish men and Italians. This is a fallacy against which those who use statistics must be continually on their guard. In very many cases age comes in as a disturbing factor. Scores of instances might be given. Why is the death rate so high among bank presidents? Not because the occupation is hazardous, but because this position is commonly meted out only to men of mature years. By the same token the average age at death of bank presidents is said to be high. The average age at death of lady stenographers is low. Why? Not because the use of shorthand and the hammering of the keys tends to premature death, but because so large a percentage of lady stenographers marry and take themselves out of the group of stenographers before they attain an advanced age. To a very considerable extent classification by occupation results automatically in an age grouping. For example, the average death rate of newsboys is low. Why? Because all newsboys are young. The average death rate of civil war veterans is high. Why? Because the entire class is now composed of old men. Likewise, certain diseases are most commonly associated with certain age periods, and some diseases are confined exclusively to one sex.

Hence, in tabulating data and in computing rates for the purpose of measuring the importance of some factor one should endeavor to so arrange the classification as to leave this as the only factor that varies. They may involve a more detailed study than we are in the habit of making, and

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it is not always possible to make such a separation of the data. The general principle should, however, always be borne in mind. Often an age grouping in addition to some other classification will be sufficient.

Specific Death Rates.—The variations in the specific death rates at different age groups are illustrated by Fig. 45, in which it will be seen that the death rates are high in early infancy. They decrease rapidly and attain a minimum at the age of about 12 years, then they slowly increase

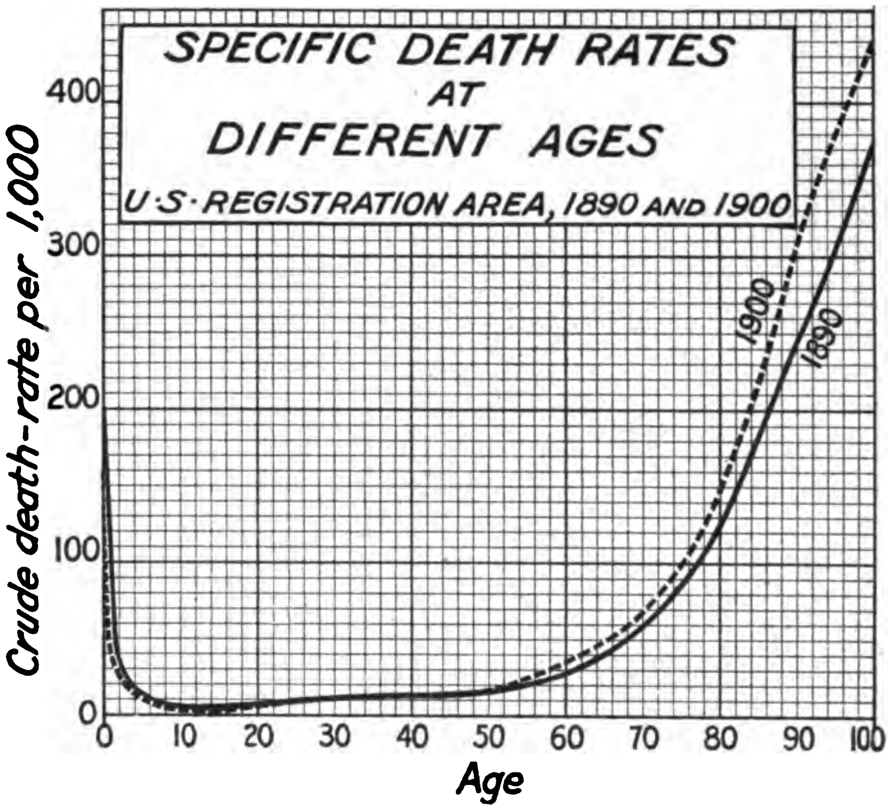

FIG. 45.

until the age of 60, and after that, of course, there is a rapid increase. Curves like this are much more satisfactory for comparing the sanitary conditions of different cities than a single figure representing the death rate can possibly be, because one may obtain at a single glance a picture of the conditions which exist at any age. The two lines in the diagram show the change that occurred in the specific death rates between 1890 and 1900 for the United States Registration Area. It will be seen that there was a decrease in the rates for infants and children, but an increase in the specific death rate above the age of 60.

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Figure 46 is equally interesting. It shows the distribution of deaths from all causes among different ages in Massachusetts in 1910. The importance of the infant mortality as compared with the deaths at other ages is strikingly evident.[1]

Display of Statistical Data.—Having made an accurate and sufficiently complete enumeration and classification of the data and compared them in a logical manner, guarding against the many fallacies that lie in wait to catch the unwary one thing yet remains—that of displaying the data so that he who runs may read, or as the farmer said when he put up a sign to beware of the dog, "so that he who reads may run." One reason why statistics produce

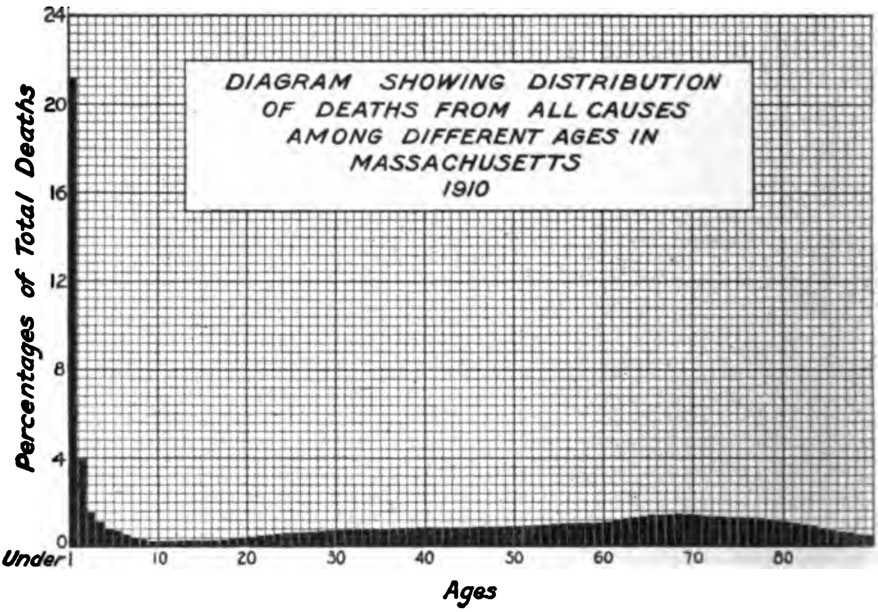

FIG. 46.

so little effect on the ordinary reader is because they are so poorly displayed. Even in tabulating data or drawing diagrams to illustrate the data graphically it is possible to falsify the facts. The art of statistical graphics has been learned by few and practised by a still smaller number of vital statisticians. And the public has become so in the habit of seeing diagrams that conceal and mislead rather than those that clearly display that it has no taste for diagrams that are really well drawn.

Tabular printing is expensive; hence, tables should be made as simple as possible, with short clear headings for the columns, good alignment, with columns numbered for convenient reference, and above all with a title that

[1] Diagrams were drawn by Mr. Sylvester Schnattschneider, Assistant in Sanitary Engineering, Harvard Uuiversity.

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tells what the table contains. The figures should not be allowed to sprawl over a page. Where there are but two or three columns these may be repeated in parallel series, unless a single set of columns is needed for the sake of presenting a clearer mental picture. Hundreds of dollars are wasted every year by using poorly arranged tables, with no one the gainer but the printer and the paper maker.

The Future of Demography.—Demography is at once a very old and a very new study. It might well be called one of the lost arts. Some of the reports that were published a half century or more ago might almost be taken as models of good statistical work. An excellent example is the report of the Massachusetts Sanitary Commission of 1850, which was prepared by Lemuel Shattuck. The vast accumulation of data in recent years has not been accompanied by an improvement in the art of classifying and studying the data. In fact there has been of late something of a retrogression. A very large number of poorly prepared reports containing undigested data have been issued. The statistical work has been crude and slipshod and has tended to bring the science into disrepute. But there are signs of a change for the better. In this movement the Bureau of the Census of the United States has been a conspicuous leader.

Aside from the academic value of the science of demography there are immense practical values which are just beginning to be realized. For example, we have come to learn that various sanitary reforms are tending to increase the cost of living. Pure food, pure air, and pure water are not obtained without expense. Oftentimes the expense is fully justified and the investment yields a substantial reward, but there are fields of activity where the rewards are less and where the increased cost of sanitation fails to yield a sufficient return. The law of diminishing returns has its application in sanitation as well as in economics. It is only by careful study of the results of sanitation that we shall be able to properly evaluate the different sanitary measures that are available to us, and it is in this direction that the practical value of vital statistics lies. More and more is the science of demography playing an important part in the administration of public health and it seems certain that at no distant future the science of demography will rank as one of the most necessary subjects for students to pursue in their training for the public health service.

It is recognized also that the realm of demography cannot be confined to the statistics of marriages, births, and deaths, but must be extended to include physiological data as well as social and economic statistics.

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DIVISION III

Legislation and Governmental Study for the Prevention of Occupational Diseases

CHAPTER I

METHODS AND SCOPE OF LEGISLATION

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In the development of our civilization from the individualism of primitive savagery to the complexity of modern industrial society, we have created many perplexing social problems. When individuals worked singly and in a very real sense "minded their own business," it was possible for the individual worker to adjust himself or his surroundings to suit at least his own best judgment concerning personally agreeable and healthful conditions of temperature, humidity, fresh air and physical strain. But when factories were built and large numbers of people came to work under artificial conditions over which they themselves had little or no direct control, there was created a very different problem. Conditions of temperature and humidity and purity of air and labor strain are now often prescribed by the most convenient, the most rapid, and the cheapest process of manufacture from the standpoint of selfish or thoughtless employers.

Every person in the community is concerned in the conservation of the health, vitality, energy, and industrial efficiency of wage earners. All agencies in society should coöperate in preventing unnecessary suffering and death among the producing classes. The protection of the health of the industrial worker is not an individual question. It is a social question demanding social regulation.

The prevention of occupational diseases is, moreover, too great an undertaking to be left entirely to individual action. It cannot be left to the worker who, even when not ignorant of the danger, is driven by necessity to his task. It cannot be entrusted to the employer whose principal business, after all, under competitive conditions, is to secure profits. It cannot be left to medical treatment alone, for prevention and not after-care is the remedy. Not only on account of the magnitude of the problem, but also because of its nature, *the prevention of occupational diseases is properly a function of government.* Governmental action, however, in promoting industrial hygiene does not mean that private or voluntary efforts of the workmen, or of their employers, or of their physicians shall be in any way minimized. On the contrary, such voluntary efforts will be vastly encouraged.

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It must be obvious to any thoughtful person that in preventing unnecessary occupational diseases we must have investigation, education and administration. These three requisites can, in a democracy, best be secured through legislation.

Investigation is expensive. But through legislation we can set aside ample funds for scientific study. In New York state during the 2 years 1912–1913 the legislature appropriated $110,000 to the factory investigating commission, in addition to the usual sustaining funds for the permanent departments of health and labor, and other states have made similar outlays.

Education is expensive. But through legislation we can provide the means for the necessary dissemination of facts gained by investigation. The reports of the occupational disease commission in Illinois and of the factory investigating commission in New York, as well as the splendid researches of several governmental commissions in England and elsewhere, have given a remarkable impetus to the task of providing sanitary work places. Reports on occupational diseases issued by our bureaus of labor have stimulated wide interest among manufacturers and wage earners. The laws in 16 states and in several European countries requiring the reporting of occupational diseases have pointed out the sore spots in industry and have resulted in valuable coöperation between physicians and factory inspectors.

Administration is essential. Protective devices often involve the expenditure of large sums of money and, under conditions of sharp competition and cheap labor in an industry, the humane and careful employer may be placed at an unfair disadvantage in dealing with his less conscientious business competitor unless there is uniform enforcement of sanitary regulations. It is only through uniformity of regulation, which legal enactments alone can secure, that the more progressive and humane employers can themselves be protected from less scrupulous competitors who would otherwise often fail to provide expensive safeguards, and it is only through compulsory regulations that the health of the employees of these unscrupulous competitors can be protected. A notable example of this was the condition which culminated successfully in the poisonous phosphorus prohibition act described in an earlier section. Match manufacturers representing 95 per cent. of the total product testified before Congress that they could not substitute a harmless compound for the slightly cheaper poison without a uniform law compelling all employers in that industry to abandon the poison. All of the other match manufacturers, however, representing the remaining 5 per cent. of the product, stood out stoutly to the last, even declaring that they would close their factories before they would submit to this sanitary measure, already in compulsory operation in practically all the civilized countries of the world. It required labor legislation to end the use of this unnecessary deadly poison before "phossy jaw," the most loathsome of all industrial diseases, could be abolished.

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Three Principal Methods.—The principal legislative remedies for the prevention of occupational diseases are of three classes. These are:

1. The method of prohibition.
2. The method of regulation.
3. The method of compensation or insurance.

The mass of legislation which falls into these three categories is constantly expanding and undergoing amendment. Any study of it is further complicated by the rapid spread, in America as well as in Europe, of the method of enforcement by administrative order, in which the legislature lays down a general rule, such as "the work places shall be safe," and leaves to a commission or to an administrative official the duty of making the necessary specific regulations. In the pages which follow the attempt has been made to present only the broad outstanding principles of the law. Much that would be essential to the legislative draftsman has been omitted in the hope that, unencumbered by excessive detail, the lay reader may arrive at a comprehensive understanding of what the various governments are doing for the abatement of occupational diseases among their wage-earning population. Full texts of the European laws cited may be found in the files of the monthly Bulletin of the International Labor Office.

1. THE METHOD OF PROHIBITION

No more striking example of the method of prohibition could be found than the suppression of white phosphorus in the match industry. As has been shown elsewhere in this volume, every leading country of the world has now outlawed this occupational poison, even resorting to the unique expedient of forming an international treaty to prohibit an occupational disease.

The only other substance against which such drastic action has been taken in industry is lead, and in this case the prohibitory legislation is confined to Europe. Austria was first to act, forbidding in 1908 the use of lead in all paints, colors or cement used for interior work, and the same year the various Swiss administrative departments were ordered to prohibit the use of white lead in painting carried on in their behalf. In 1909, however, France took the most advanced stand by declaring that after July 20, 1914, the use of "white lead, of linseed-oil mixed with lead, and of all products containing white lead will be forbidden in all painting, no matter of what nature, carried on by working painters either on the outside or in the inside of buildings." Whether such prohibition is to spread depends largely upon the success of other methods in combating plumbism.

Another type of prohibitive labor legislation is directed against the use of dangerous instruments. Thus in textile mills the extent to which tuberculosis and other contagious diseases were transmitted by the workers' sucking thread into the shuttles led to this process being called the "kiss of death."

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In 1911, accordingly, Massachusetts forbade the use of any form of shuttle which requires the employee to use his lips or mouth in threading it.

2. THE METHOD OF REGULATION

The method of regulation, in the prevention of occupational diseases as in other social problems, is based on the principle of toleration within limits. The majority of the people may believe that certain harmful conditions are so necessary a part of our industrial life that their prohibition is at present undesirable or at least impracticable. As a result, the method of regulation is far more widely used than the method of prohibition, as well as being historically much older.

The adoption of this method leads to limitations in three different directions: (1) upon the methods of handling the hazardous material; (2) upon the period of exposure; and (3) upon the persons exposed. These three types of limitation are seldom used singly, but are usually combined in the same law. In the following discussion no attempt will be made to describe the laws now existing in most industrial states and countries with reference to air-space, ventilation, general dust and fume removal, temperature regulation, toilet facilities, weekly rest days, and similar general matters, because while all these measures have a bearing on the health of the operatives they are not designed as protection against any specific occupational illness. No mention will be made, either, of provisions applying only to the labor of women and children, as such provisions are treated separately elsewhere in this volume.

Lead.—The most widespread of industrial poisons, lead is also most frequently the subject of regulative laws. Public attention was first called to its dangers in America by the report of the Illinois Commission on Occupational Diseases in 1911, since which date six states have enacted specific protective measures.

The most advanced of these measures, prepared by the American Association for Labor Legislation, is that adopted in practically identical form by the Ohio and Pennsylvania legislatures in 1913, and in New Jersey in 1914. Every employer is required to provide, without cost to his workmen, reasonably effective devices to prevent illness incident to the processes at which they are engaged. In addition, specific regulations are made to protect those employed in the manufacture of white lead, red lead, litharge, sugar of lead, arsenate of lead, lead chromate, lead sulphate, lead nitrate or fluo-silicate and, in the New Jersey law, in the manufacture of pottery, tiles, or porcelain enameled sanitary ware. Workrooms must be thoroughly lighted and ventilated. Separate rooms must be provided for the dusty and therefore more dangerous processes, with floors which can be cleaned daily either by wet methods or by vacuum cleaners. Vessels for crushing, drying or conveying the lead or lead salts must be protected by hoods or dust removers. Great emphasis is laid on washing facilities, the requirements including hot and cold water, soap, towels, nail brushes, and shower baths;

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there must also be separate dressing rooms, eating rooms, drinking fountains, and double lockers, and respirators must be furnished. At least 10 minutes on the employer's time must be allowed each employee before lunch and at closing for the use of the wash room, and an additional 10 minutes twice a week for the use of the shower baths. The employees are made subject to fine in case of failure to use the facilities for cleanliness which are provided, and are not to take any food or drink into the workrooms. Every employee exposed to danger from lead dust or fume must be examined medically at least once a month by a physician paid by the employer. If lead poisoning symptoms are found, a record must be filed with the factory inspection department within 48 hours and a copy sent to the employer, who may not thereafter employ that worker in any place exposing him to lead without a written permit from a physician. Notices in English and in such other languages as the circumstances require must be prominently posted, stating the dangers and giving instructions for avoiding them.

The Missouri law, also of 1913, and the Illinois act of 1911 contain very nearly the same provisions with respect to washing and lunch-room facilities, dust removal, and medical examination of employees. They lack, however, the clause imposing hygienic duties on the workers as well as on the employers. Both these laws, also, differ from that just described by being less specific but more inclusive. The Illinois act of 1911 applies, in addition to occupations involving contact with the familiar poisonous lead salts, to brass manufacture and to lead and zinc smelting, while the Missouri statute aims to cover any process "in which antimony, arsenic, brass, copper, lead, mercury, phosphorus, zinc, their alloys or salts, or any poisonous chemicals, minerals, acids, fumes, vapors, gases or other substances are generated or used in harmful quantities or under harmful conditions."

The New York law, enacted in 1912, merely forbids the taking of food into any workroom where lead, arsenic or other poisonous substances are present, and requires the maintenance of a separate lunch room.

In Europe the tendency is away from such omnibus legislation and toward minute regulation for each of the important lead industries separately. This tendency is perhaps best exhibited in Germany, which country not only was one of the first to establish scientific standards for the lead-using trades, but has to-day the greatest mass of legislation bearing on the subject. Separate statutes exist for lead mining, casting, zinc smelting, type founding and stereotyping, typesetting, white lead manufacturing, lead oxides and paints, electric accumulators, painting and file cutting. England has passed separate laws or issued separate administrative orders governing lead smelting and casting, lead founding, white lead, lead oxides and paints, electric accumulators and file cutting, heading yarn dyed with a lead compound, earthenware and china, color transfers on earthenware and china, and tinning and enameling. In Austria, France and Belgium similar but shorter lists of industries have been individually regulated.

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In the more dangerous trades in the countries named, the workers are forbidden to eat or to leave the premises where they are employed until they have first thoroughly washed their hands and their faces and in some cases their mouths and noses also. No food or beverage of any kind, and no tobacco either for smoking, chewing, or snuffing, may be used in or even carried into the workroom. In the more dusty trades, such as white lead making, lead oxidizing and electric accumulator manufacture, the men must take warm baths regularly, in some cases weekly and in some cases daily. In the white lead industry in England a register must be kept of these baths.

Much emphasis is thrown on adequate ventilation and lighting. The walls and floors of the chambers where dusty processes are carried on must be hard and impervious, and must be cleaned by moist methods or with a hose at regular intervals. Mechanical, hooded contrivances for crushing, mixing and transporting dangerous lead products are generally specified in the law. The employer must furnish working clothing, gloves, respirators, and sometimes head coverings, and lockers in which to keep them. There must be dressing rooms, and wash rooms supplied with hot and cold water, soap, towels, nail brushes, and bathing facilities; a dust-free room, warmed in cold weather and in some cases provided with means for warming the workmen's food, must be set aside as a lunch room. In some of the more arduous trades the hours are made short and are broken by frequent rest pauses. Notices of the law must be posted, and sometimes must also be distributed to the employees.

Germany in the lead smelting, white lead, lead oxide, storage battery and painting trades, England in all but the last of these, with the addition of yarn heading and earthenware, and Austria in lead and zinc smelting require a "control book" or health register. The German book is a very elaborate affair, requiring records of the name of the person keeping it, first and last name, address and age of each workman, date of his entering and leaving the employment, date and nature of his illness, date of his recovery, name of the factory physician, and dates and results of the medical examinations. The employer is responsible for the correctness of this record and must show it to the factory or medical inspector on demand. The Austrian health register goes into even more detail.

Before a workman in Germany, England, France, Austria, or Russia can legally obtain work in the dangerous lead trades, he must provide himself with a medical certificate showing that he is of sound physique and constitution and fairly capable of withstanding the poisons with which he will have to work. Lung, kidney or stomach trouble, alcoholism and a generally weak constitution are debarring ailments in Germany. The French law excludes workers who exhibit symptoms of lead poisoning or of any complaint likely to be dangerously aggravated by plumbism. In Belgium no workman addicted to alcohol may be employed in white lead, lead oxide or lead paint making.

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Having secured employment in a lead trade, the workman's continuation therein is usually dependent on his successfully resisting its inherent hazards. The frequency of compulsory medical examination ranges from 1 every 6 months for painters in Germany to 1 a week in the English white lead industry. In most cases, however, 1 examination a month is the requirement; in white lead and lead oxides in Germany it is 2 a month. When a worker is discovered to have symptoms of plumbism, the laws almost universally require that he be "suspended," that is, given employment which keeps him out of contact with lead until he has fully recovered. Belgium, in the white lead industry, requires that a leaded man be kept out of that sort of work permanently.

A decisive clause found repeatedly in the German laws, and also in the Austrian statute on lead and zinc smelting, is that a workman continually violating, after warning, the hygienic duties laid upon him is to be permanently discharged without notice. A similar but much weaker clause is the provision occasionally found in England that a reckless worker lays himself open upon conviction to a fine.

Several countries have turned their attention to house painting, a prolific source of painful and annoying but less often fatal attacks of plumbism. France, Germany and Belgium forbid all rubbing or scraping of lead paint in a dry state. In France the manipulation with the bare hands of any material containing white lead is also forbidden. Belgium in 1909 decided that white lead for painting could be transported, sold or used only in the form of a paste or liquid mixed with oil. The ministry was given power to extend the same restriction to white lead for any other purpose, but later the law was weakened to allow special permits to persons who could prove the necessity of their using white lead in powder or cakes. A mild ministerial order is found in Austria, where it is allowable to import or sell lead colors or cement only if the fact that they contain lead is "expressly marked upon them in a clear and comprehensible manner."

Brass.—Legislative provision is made for the control of brass fume and dust under the Illinois and Missouri lead laws already mentioned, but nowhere else specifically in this country. In England exhaust drafts for the prompt removal of fumes, and arrangements for preventing the fumes from spreading to other rooms, must be provided. For the personal care of the employees, also, there must be washing accommodations, with soap, nail brushes and towels.

Miscellaneous Dusts.—Three states—Illinois, Michigan and New Jersey—have detailed legislation looking toward the removal of the miscellaneous dusts generated in the grinding and polishing rooms of many industrial establishments. These laws designate the kind of machines covered by the act, how the protective devices shall be adjusted to the machines, the dimensions of the connecting pipes, and the degree of the angles as well as the rate of speed at which the fans or blowers shall run. In addition, about half

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the states in the union have enacted more general laws on the same topic. Fairly representative of this type of legislation is the Oregon statute of 1907, providing that "if in any factory, mill or workshop any process is carried on in any enclosed room thereof, by which dust is generated and inhaled to an injurious degree by the persons employed therein, conveyors, receptacles or exhaust fans, or other mechanical means, shall be provided and maintained for the purpose of carrying off and receiving such dust." As scientific standards of dust control and removal are developed, and inspectors become technical experts and advisers instead of mere detectives, such laws can be made more and more useful agencies in factory hygiene.

Similar general legislation, directed mainly toward the protection of metal polishers, exists in a number of European countries, among them being Holland, Italy, and Norway.

Compressed Air.—Another important source of industrial illness and death which has been brought under legislative regulation in America is work in compressed air. The earliest American statute designed to combat this hazard is the New York act of 1909, which has twice been strengthened by amendment and is now much excelled by the New Jersey law of 1914.

The New Jersey law requires a medical examination of all persons applying for employment in caissons or tunnels where work in abnormal air pressure is carried on. Only those found physically qualified may be employed, and persons addicted to the excessive use of intoxicants are expressly debarred. Examinations must be repeated on returning to work after 10 days' absence and after 3 months' continuous employment, and records must be kept of all examinations.

Working periods and rest intervals are carefully regulated in accordance with the following scale, in which "pounds" refers to pressure in addition to the normal atmospheric pressure of 15 lb. to the square inch. No work is allowed in pressure over 50 lb. except in cases of emergency.

If the pressure exceeds, pounds	But does not exceed, pounds	Number of hours work in 24, hours	Interval between working periods, hours
Normal	21	8	½
21	30	6	1
30	35	4	2
35	40	3	3
40	45	2	4
45	50	1½	5

As too rapid emergence from high pressure is the main cause of caisson disease, a decompression lock is required, through which the workman must pass as he leaves the work, and the speed of decompression is held within safe limits. In tunnels decompression may be at the rate of 3 lb. every 2 minutes, unless the pressure is over 36 lbs., when it may be only 1 lb. a minute. In caissons a more rapid rate is permitted, to wit:

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If the pressure exceeds, pounds	But does not exceed, pounds	Time required for compression, minutes
Normal	10	1
10	15	2
15	20	5
20	25	10
25	30	12
30	36	15
36	40	20
40	50	25

Dressing rooms with hot and cold shower baths must be kept open during and between working periods, and also a separate room for drying clothes. If the pressure exceeds 17 lb. a double medical lock is required, in which sufferers from "the bends" may be placed for recompression and treatment, and which must be equipped with inside and outside air gauges, time pieces and a telephone. All equipment must be inspected every day. One or more licensed physicians must be present at all necessary times, and if the pressure exceeds 17 lb. the constant attendance of registered nurses or of other competent persons certified by the medical officer is also required.

France and Holland have also legislated concerning compressed-air work. The French law requires from applicants medical certificates of fitness, which are to be renewed 2 weeks later and subsequently every month, and establishes minute regulations governing safety appliances, medical locks, and time of decompression. In Holland the supervising physician is appointed and paid by the government, and must give the workmen necessary instructions with regard to health. A long list of ailments is included in the law, any of which debar from work in compressed air. These ailments are malformation, imperfect physical development, general debility, obesity, chronic diseases of the skin, inflamed or swollen glands, boils or abscesses which may be expected to cause trouble, excessive perspiration of the feet, chronic affections of the bones or of the muscular system or joints, diseases of the heart or of the nervous system or the blood, affections of the trachea or lungs, chronic affections of the digestive organs, rupture, diseases of the kidneys or bladder, gonorrhea, suspected alcoholism and affections of the nose and ears.

The use of the diving dress in sponge fishing has been prohibited in Austria-Hungary, Crete, Egypt, and Samos, while both Greece and Italy have placed restrictions on the depth to which divers may go, the Grecian limit being 127 ft.

Textile Dust.—Specific regulations against dust and lint in textile establishments have for some years been established in England. In the dusty rooms exhausts and respirators are required, as well as thermometers and hygrometers for the regulation of temperature and humidity. The law provides also for the purity of the water used for humidifying. These regula-

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tions apply to the spinning and weaving of flax, tow, hemp and jute. In Massachusetts a 1910 law safeguards the purity of the water used in humidifying weaving and spinning rooms, and standardizes the thermometers for regulating temperature.

Rag Dust.—An Austrian law aims to protect workers in paper mills from the irritating and infectious dust often carried by old rags. Tearing and dusting of the rags must be done mechanically. Washing, bathing and dressing accommodations, working clothes, respirators and first-aid materials are required, and eating and smoking are prohibited in the workrooms. Persons with delicate respiratory organs, consumptives, and workers with open wounds may not be employed.

Stone Dust.—To guard the workers in stone quarries and stone-dressing establishments from the dangerous dust created, Germany requires that the floors shall be kept damp, and workmen may not sit closer together than 2 meters (about 6½ ft.). Roof shelters must be erected to protect the workers from the weather; men may work at dressing sandstone only 9 hours a day, and at other occupations 10 hours. In Austria quarrymen must be provided with goggles or eye shades. The regulations in Holland are more thorough. Hours are limited to 10 a day for the first 2 years and 9 thereafter, precautions must be taken against the formation and spread of dust, and workmen are entitled to medical examination once a year.

Chromates.—Provisions for the prevention of chrome ulceration and other diseases caused by contact with chromates are found in Austria, Germany and England. The German rules are that dust-producing processes must be carried on away from the general workrooms, and the chromates must as far as possible be crushed in hermetically sealed apparatus. Working clothes, caps and respirators are required for the employees, together with warm baths, soap, towels and nail brushes, cloak rooms and lunch rooms. For employment a certificate is necessary, stating that the applicant has no open wounds or skin disease. Monthly medical examinations are required, and workmen showing symptoms of chrome poisoning in the form of irritation of the mucous membrane of the nose must be suspended.

The English regulations covering the manufacture of chromates of sodium are almost identical with the German. A notable addition is that foremen must report to their managers the names of workmen who fail to observe the rules for personal hygiene.

Bisulphide of Carbon.—In rooms where bisulphide of carbon is used in vulcanizing rubber, England forbids work for longer than 5 hours a day, and no food may be eaten there. Employees must be examined monthly, and records kept of the examinations.

More detailed precautions against the same poison are established in Germany. Provision is made for special ventilators, devices to prevent the escape of the fumes, cloak rooms and washing accommodations with soap and towels. Monthly medical examinations are required.

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Mercury.—In animal hair-cutting establishments where mercury is used, France requires the posting of the following notice:

Notice.—Mercury and its compounds are dangerous. They may enter the body with the air breathed in (dust, vapors), with food (unclean hands, unclean tables), through the skin (cracks, scratches, or cuts).

Should you have any cracks, scratches or cuts please inform the management immediately of the fact.

Before eating or drinking carefully clean your hands with soap and your mouth with drinking water.

Should you have any pains in the mouth or teeth and excessive quantity of saliva, should you shiver, should you have swellings of the legs, the hands, or under the eyes, consult the doctor at once.

Tobacco.—Injurious quantities of tobacco dust are carefully guarded against in Germany. The floors, ceiling, height of walls, windows and air-space of rooms where tobacco is worked up are regulated. The tobacco must be kept moist, and only enough for 1 day's work may be stored in the work-room, which must be aired three times a day and thoroughly cleaned twice a year. The dust must be removed from floors and tables daily by washing or by damp duster, and spittoons and lavatory accommodations with soap and towels must be provided. Spitting on the floor is prohibited, the mouth must not be used in making cigars, nor may the cigar knife be moistened with saliva. A worker who violates the rules after repeated warning is to be discharged without notice.

Anthrax.—Of the infectious diseases which have gained occupational prominence, anthrax—described in detail in an earlier chapter—has most widely been made the subject of preventive legislation Austria, Belgium, France, Germany, some of the German kingdoms, Great Britain and Italy have all turned their attention to stamping out the disease; the United States still lags behind.

In 1901 Great Britain ordered that provision be made for keeping the workers' food and clothing in a clean place. Water, soap, towels and nail brushes are to be furnished, and all requisites for treating scratches and slight wounds must be at hand. If dangerous hides are handled, overalls protecting the neck and arms, and gloves, are required. It is the duty of every workman having an open cut to report the fact immediately to the foreman and to withdraw from the shop until the cut is healed. Still more careful regulations were later prescribed for work on horsehair from China, Siberia or Russia, and on wool, goat and camel hair. In addition to the foregoing requirements, material which has not been disinfected must be kept in a separate room to which no food, drink or tobacco may be taken, and no work except opening and sorting may be done upon it before disinfection. In addition to overalls and head coverings, the work-people must be provided with respirators.

The German rules for disinfection and for sanitation of the work place are

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more strict, but otherwise the regulations follow very closely those of England. An administrative order in France, dated October 1, 1913, compiles and amplifies several earlier rules. Three notable requirements are the appointment of a factory physician for the examination, treatment and recording of cases, a first-aid box, and lime-washing of the workrooms "whenever necessary, and especially when a case of anthrax has occurred."

Belgium first turned its attention to the prevention of anthrax among 'longshoremen, forbidding in 1903 their direct touching of skins, horns, bristles or other material capable of conveying the infection, and dock workers with wounds on exposed parts of the body were required to bandage the wounds before handling such material. Five years later managers of brush factories were ordered to subject all bristles, immediately upon unpacking, to a system of disinfection capable of destroying anthrax spores. Austria in 1913 enacted the sweeping provision that on the appearance of any one of a number of contagious diseases, anthrax among them, a plant might be ordered closed if its operation entailed urgent danger to the population. Italy has as yet gone no further than offering a prize for the best essay on anthrax prevention.

Ankylostomiasis.—As the infection of miners with ankylostomiasis, or "miners' hookworm," takes place mainly through soil pollution by others already suffering from the parasite, a few countries have taken precautions to prevent persons with the disease from securing employment in mines. In 1904 the Austrian Minister of Agriculture issued a circular to the mine inspectors in the districts of Vienna, Klagenfurt and Kracow prescribing the examination of applicants with this end in view. Similar laws exist in the Dortmund district of Prussia and in the kingdom of Saxony, while a royal order prescribing this and other hygienic precautions was issued in Spain in 1912.

Contagious Diseases among Glass-blowers.—While many industrial conditions predispose to tuberculosis, in no case is the occupational causation of this disease clearer than when it occurs among glass-blowers. A French law requires the appointment of a physician for the medical supervision of employees in glass works, and the keeping of a health register. The same law contains the rather weak provision that "workers shall not be allowed to undertake any work necessitating the common use of one blowpipe, except only by the written certificate of the medical man to the effect that they are not suffering from any contagious disease at a stage in which such illness is capable of being transmitted by the blowpipe." An earlier law in Portugal makes the prohibition complete. It says: "As good ground exists for supposing that the prevalence of tuberculosis in glass-blowing works is largely caused by permitting free use of the same blowpipe by different workmen, his majesty the king hereby commands that every workman shall be provided with a blowpipe for his personal use, marked with his name or number, and that the use of such blowpipe by other workmen shall be absolutely forbidden."

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3. THE METHOD OF COMPENSATION OR INSURANCE

The third and last of the principal legislative remedies for the prevention of occupational diseases is the method of compensation or insurance. While newer than the other two methods, its results are already by no means inconsiderable.

The method of compensation or insurance is based on the principle that those individual misfortunes which cannot be prevented, either by prohibition or regulation, can best be borne by the community. When applied to industrial injuries sustained by workmen in the course of their employment, the justification for this principle is particularly obvious. It is worthy of note also that with almost startling rapidity the American people are coming to demand that workingmen's insurance be recognized as a proper function of government.

The expense of broken machines is borne by the industry, which passes the burden on to society. In recent years we have come to insist that so, also, should be borne the financial expense of broken legs and arms. The next step must be the extension of the same principle to include incapacity due to arms paralyzed by lead poisoning or incapacity due to other diseases of occupation. Instead of leaving the money loss, as well as the physical suffering, to be borne by the unfortunate victim or his family, the financial burden of relief should be placed upon industry where it belongs.

During the past few years the industrial accident problem, under the urge of impending legislation, has attracted the abilities of hundreds of experts. In addition to stringent regulations for the direct prevention of such industrial injuries, 33 states, in their efforts to provide adequate relief for the victims, have boldly overthrown the discredited and out-worn system of employers' liability and have adopted the workmen's compensation or insurance principle. No agitation has been more effective in preventing accidents than the campaign to provide for the victims a just system of indemnity.

And in attempting to deal effectively with the occupational disease problem, practical people will very soon recognize the wisdom of placing upon those responsible for unhealthful work conditions the financial burden of caring for the victims. No intelligent person can go far in the study of compensation for industrial accidents without realizing that a logical consideration of the facts must lead likewise to compensation for industrial diseases. In fact the arguments used so effectively by advocates of compensation for accidents, and now so generally accepted by all men, apply with even greater force in the consideration of relief for the victims of occupational diseases. A considerable part of the money now paid to employers' liability companies and to ambulance chasers could, under a just system of compensation, go where it belongs—to the injured workman or his family. Expensive, annoying, and unsatisfactory litigation could be reduced to a minimum. Information concerning special danger points in industry would be automatically pointed out to the factory inspectors in a manner both prompt and sure.

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Unnecessary occupational diseases would then be prevented, and that is the real problem.

While a few large awards have been made under liability laws, American victims of occupational diseases are still, except in California and Massachusetts, practically without relief. In the latter state, beginning in 1914, the supreme court has several times upheld awards by the industrial accident board on the ground that the occupational diseases compensated were "personal injuries" within the meaning of the act. The California legislature in 1915 brought occupational diseases within the scope of her compensation law of 1911 by striking out the word "accidental" as applied to compensable personal injuries.

The United States government, since 1908, has had an accident compensation law applying to certain civilian employees. But victims of occupational disease in the service of the country are excluded from its benefits. In 1913 the Department of Commerce published a report upon the operation of this law. Sixty-six closely printed pages are devoted to embarrassing questions arising out of occupational diseases contracted in the government service. One of the most urgent recommendations for a change in the law is that it be extended specifically to embrace diseases of occupation, and a carefully drafted bill for that purpose has been introduced into Congress at the instance of the Association for Labor Legislation. When this measure is enacted, the United States will have a compensation law which is abreast of those of the most progressive nations of the world.

Great Britain.—The country which led and still leads in the matter of compensation for occupational diseases is Great Britain. Its workmen's compensation act of 1906 contained a pioneer schedule of six diseases for which compensation was to be paid on the same basis as for accidents. This list has twice been extended until to-day no fewer than 25 maladies of occupation entitle the victims to relief. So important is this list that it is given in full:

OCCUPATIONAL DISEASES SUBJECT TO COMPENSATION UNDER THE BRITISH WORKMEN'S COMPENSATION ACT

Diseases Listed in Original Act, December 21, 1906

Description of disease	Description of process
Anthrax.	Handling of wool, hair, bristles, hides and skins.
Lead poisoning or its sequelæ.	Any process involving the use of lead or its preparations or compounds.
Mercury poisoning or its sequelæ.	Any process involving the use of mercury or its preparations or compounds.
Phosphorus poisoning or its sequelæ.	Any process involving the use of phosphorus or its preparations or compounds.
Arsenic poisoning or its sequelæ.	Any process involving the use of arsenic or its preparations or compounds.
Ankylostomiasis.	Mining.

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DISEASES ADDED BY ORDER OF SECRETARY OF STATE, MAY 22, 1907, DECEMBER 2, 1908, AND JULY 30, 1913

Description of disease or injury	Description of process
1. Poisoning by nitro and amido derivatives of benzene (dinitrobenzol, aniline, and others), or its sequelæ.	Any process involving the use of a nitro or amido derivative of benzene or its preparations or compounds.
2. Poisoning by carbon bisulphide or its sequelæ:	Any process involving the use of carbon bisulphide or its preparations or compounds.
3. Poisoning by nitrous fumes or its sequelæ.	Any process in which nitrous fumes are evolved.
4. Poisoning by nickel carbonyl or its sequelæ.	Any process in which nickel carbonyl gas is evolved.
5. Arsenic poisoning or its sequelæ.	Handling of arsenic or its preparations or compounds.
6. Lead poisoning or its sequelæ.	Handling of lead or its preparations or compounds.
7. Poisoning by Gonioma Kamassi (African boxwood) or its sequelæ.	Any process in the manufacture of articles from Gonioma Kamassi (African boxwood).
8. Chrome ulceration or its sequelæ.	Any process involving the use of chromic acid or bichromate of ammonium, potassium, or sodium, or their preparations.
9. Eczematous ulceration of the skin produced by dust or liquids, or ulceration of the mucous membrane of the nose or mouth produced by dust.	
10. Epitheliomatous cancer or ulceration of the skin or of the corneal surface of the eye, due to pitch, tar or tarry compounds.	Handling or use of pitch, tar, or tarry compounds.
11. Scrotal epithelioma (chimney-sweep's cancer).	Chimney-sweeping.
12. The disease known as miners' nystagmus, whether occurring in miners or others, and whether the symptom of oscillation of the eyeballs be present or not.	Mining.
13. Glanders.	Care of any equine animal suffering from glanders, handling the carcass of such animal.
14. Compressed air illness or its sequelæ.	Any process carried on in compressed air.
15. Subcutaneous cellulitis of the hand (beat hand).	Mining.
16. Subcutaneous cellulitis over the patella (miners' beat knee).	Mining.
17. Acute bursitis over the elbow (miners' beat elbow).	Mining.
18. Inflammation of the synovial lining of the wrist-joint and tendon sheaths.	Mining.
19. Cataract in glass workers.	Processes in the manufacture of glass involving exposure to the glare of molten glass.
20. Telegraphist's cramp.	Use of telegraphic instruments.
21. Writer's cramp.	

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This apparently makes a list of 27 diseases subject to compensation. It will be noted, however, that two of these (lead poisoning and arsenic poisoning among the diseases added by the secretary of state) are duplications, inserted merely to widen the scope of occupations to which the act applies, the "Description of Process" in both cases being changed to include the "handling" as well as the "use" of the dangerous material.

Under the British law expenses of medical attendance and burial up to £10 are paid in cases of death if there are no dependents. If there are, they receive a maximum sum equal to 3 years' earnings, with minimum and maximum limits of £150 and £300. In case of total incapacity, 50 per cent. of wages is paid, not exceeding £1 weekly, while for partial incapacity the injured workman may receive the entire difference between his earnings before and after illness.

South Australia.—The precedent set by Great Britain in 1906 has been followed by two of her colonies. Five years after the passage of the British law South Australia repealed its earlier compensation code and adopted a new one, scheduling for compensation the same six diseases with which occupational disease indemnity had begun in the mother country. The rates of compensation are, however, considerably higher than in Great Britain. Burial expenses are increased to £20, and the minimum compensation to dependents is £200 instead of £150. For partial as well as for total disability 50 per cent. of wages, not to exceed £1 a week, is given. On the other hand, remuneration for disability, instead of running for the entire period, is limited to £300.

Ontario.—In 1914 the Canadian province of Ontario adopted its first workmen's compensation law, modeled after that of England, and scheduling the same original six diseases. Burial expenses are limited to $75, and compensation to dependents to $40 a month. Benefit for total disability is 55 per cent. of wages, and for partial disability is 55 per cent. of loss of earning power, both payable for life.

France.—In France the principle of compensation for occupational diseases is recognized, but is as yet limited in application to two highly infectious diseases. A section of the financial law of 1911 requires employers of miners suffering from ankylostomiasis to bear the expense of all medical treatment, and also to pay benefits during the treatment equal to those established under the accident compensation law—66⅔ per cent. of wages for total disability and 50 per cent. of loss of earning power for partial disability. Anthrax is also rated, for compensation purposes, as an accident.

Germany.—In Germany, also, anthrax is classed as an accident for purposes of compensation. Moreover, in Germany, as well as in many other countries, occupational diseases are to some extent taken care of under national systems of health insurance. The rates of benefit under this form of insurance are, however, much lower than under accident insurance or compensation, and consequently sentiment is steadily growing in favor of

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placing diseases directly attributable to the industry in the latter system, where they properly belong. The strength of this sentiment in Germany is revealed in the workmen's insurance code of 1911. Here it is provided that: "By decision of the federal council the accident insurance can be extended to specified occupational diseases in industries."

Switzerland.—In Switzerland the same movement is also in progress. The Swiss federal law of 1911 relating to sickness and accident insurance provides that "the federal council shall prepare a list of substances, the production or employment of which occasions dangerous diseases. Every disease exclusively or essentially due to the action of one of these substances in an enterprise subject to insurance is deemed an accident within the meaning of the present law." This provision, it will be seen, is stronger than that in the German code, which is merely permissive.

Bulgaria.—Industrial disability compensation in Bulgaria is as yet limited to employees of the state, but for them the law of 1906 establishes compensation for accident and for illness if it "arises out of" the employment.

Health Insurance.—While, as has been pointed out, the benefits to the employee under a system of health insurance are lower than under accident compensation, much may be expected, in the direction of occupational disease prevention, from the rapid spread of compulsory health insurance throughout the world. Ten countries—Austria, Germany, Great Britain, Holland, Hungary, Luxemburg, Norway, Roumania, Russia and Serbia—already have such compulsory systems, while a number of others, Switzerland, for instance, offer subsidies to voluntary health funds. An active campaign for a compulsory health insurance system is now under way, also, in the United States.

Conclusion.—At a time when the health of the people receives thoughtful attention, and sociologists, lawyers and physicians vie with one another in pressing upon national legislatures the need of action, the effects of industries upon health call for thougthful study. At a time when compensation for accidents received in employments is the most actively discussed phase of labor legislation, the question of occupational diseases demands consideration.

Gradually, and surely, but almost without realizing it, we have passed within the last 15 years into a new period of economic history. The courts are begining to get a new perspective on life. Century-old precedents based on individualism are trembling in the balance. Here and there a judge shows clearly that he has been studying economics as well as law. Recent court decisions clearly indicate that protective labor legislation will be sustained as a legitimate exercise of the police power when it is made apparent that conditions justify such interference on the grounds of health.

Future labor legislation, and court decisions which mark the path of social progress, will be based upon scientific study of industrial hygiene. Only through careful study can we secure accurate information which will finally

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enable us to establish scientific standards. The establishment of such standards is prerequisite to intelligent legislation, to enlightened court opinions, and to efficient enforcement of the law.

While such researches are under way, we should emphasize at every opportunity the following considerations: (1) all preventable occupational diseases must be prevented; (2) those occupational diseases which we do not yet know how to prevent must be reduced to a minimum; and (3) the victims of occupational disease must be compensated for their injuries by some just system of insurance.

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CHAPTER II

THE NOTIFICATION OF OCCUPATIONAL DISEASES

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For most adults labor is a necessity. Each must do the equivalent in work of the labor necessary to clothe, feed, and house himself. If he does not, some one must do it for him. Work is not only necessary, but if done under proper conditions is beneficial to the individual; it exercises, develops and trains his mind and body and makes life more pleasant. Work should be beneficial to and not destructive of the worker; it should be physiological and not pathological.

There are few industries which do not or may not cause ailments or disabilities among those engaged in them. In some the worker is merely made more susceptible to certain diseases, in others actual disease is produced unless proper precautions are taken to safeguard the employee's health. In industrial communities a considerable proportion of all diseases and illnesses among wage earners is due directly or indirectly to occupation or to conditions associated with labor. In such communities a large part of public hygiene relates to the industries.

The ever increasing complexity of our civilization and especially the increasing complexity of our industrial life, with its rapid development, its introduction of new machinery and processes, has introduced new occupations, new industrial poisons, and new occupational hazards. The conditions of labor and the environment of the worker have changed much in the last 30 years. Some of the changes have been to the worker's advantage; others have been to his detriment, physical or mental. The worker's labor should add to his welfare and be a factor in increasing his well-being. It should not be a cause of injury. Occupation ceases to be a source of profit to the worker if it takes from him not only his time and labor but his health as well and causes physical or mental injury or disease. The making of white phosphorus matches has been legislated out of existence in the United States because it crippled and mutilated men. Other industrial processes that multilate and kill will probably meet a similar fate.

The attention being given to the matter is indicated to some extent by the fact that within the last 5 years the legislatures of 15 states have enacted laws requiring physicians to report to the authorities cases of certain occupational diseases occurring in persons treated by them.

Why Cases of Occupational Disease Should be Reported.—A disease may be prevented by entirely removing the cause from the community and

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rendering it impossible for the cause to again gain entrance. Theoretically leprosy could be eliminated from a locality by removing all lepers and preventing infected individuals from again coming in. Phosphorus poisoning has been practically eliminated from the United States by the act of Congress placing a prohibitive tax upon the manufacture of white phosphorus matches. However, there are few diseases which can be prevented in this way. From the standpoint of the community most of the so-called preventable diseases must be controlled. Only a few can be absolutely eliminated.

To control a given disease a primary requisite is to know where and under what conditions cases of the disease are occurring. This is equally true, whether the disease is plague, cholera, typhoid fever, tuberculosis, lead or arsenic poisoning, or compressed-air illness, for each case shows the existence of conditions capable of producing the disease, conditions which should be remedied, so that others will not be subsequently affected from the same source. The notification of cases is as important in the control of occupational diseases as it is in the control of communicable diseases.

The notification of cases of occupational diseases will, however, have little other than statistical value unless the source of each case is investigated by some competent person. This is especially true of the industrial poisonings. The investigation of the conditions under which each case arises should lead to such changes as will prevent the infliction of similar injury to others. The accomplishment of this should be the purpose of case reports, and the desire of the community, the employer, the patient and the attending physician.

By Whom Reports Are Made.—Reports of the occurrence of occupational diseases are necessarily made by physicians, for because of their training they are as a class the only persons capable of recognizing the nature of maladies and of ascribing them to their true causes. However, the average physician is prone to fail to recognize the etiologic relation of occupation in his cases. This is due in large measure to the fact that many occupational diseases are of comparatively recent origin, being the result of industries or manufacturing processes developed mainly in the last 20 or 30 years. As a result, but little regarding them is found in the literature accessible to and used by practising physicians. Nor has the teaching of the intimate relation of occupation to the diseases found in industrial communities made its way into the curriculums of many of our medical schools. Nevertheless, the physician is the one whose training best fits him to recognize cases of industrial disease and, besides, he is the one who sees most of the sick, at least a greater proportion than any one else. It is necessary, therefore, to rely upon the medical practitioner for information as to when and where cases are occurring, and for this reason the progress made in the control and prevention of occupational diseases and in the advancement of industrial hygiene will depend largely upon his coöperation. The medical profession

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can make the control of occupational diseases a thing of early accomplishment or can retard it for a long time.

The laws of the states of Illinois, Missouri, New Jersey, Ohio, and Pennsylvania require employers to cause all employees engaged in certain harmful kinds of work or processes to be examined at least once a month to ascertain whether the nature of the work has caused injury or illness. The physicians making these examinations will undoubtedly become more and more expert and will recognize occupational morbidity with greater facility and certainty than do the physicians who see fewer cases. The examining of employees in this way will bring to attention practically all the illnesses with which they are afflicted and among these will be cases that would have otherwise never been brought to the attention of a physician.

To Whom Reports are Made.—In some states the physician reports cases of occupational disease to the state department of labor, but in most of the 15 states requiring notification the reports are sent to the state board of health. In one state they are sent both to the state board of health and to the department of labor. In several of the states in which the reports are sent to the health department, that department is required to send copies immediately to the labor office.

The chief purposes of morbidity reports, that is, reports of cases of sickness, being that each case may be known and investigated, faulty conditions remedied and the occurrence of the disease in others prevented, reports must reach without delay the officials who will investigate the reported cases and institute measures to protect others. In the notification of the ordinary communicable diseases the reports are sent to the local health officer, for he is the one who must get the information promptly and act upon it. Reports of occupational diseases are sent to the state department of labor by the physician, either directly or indirectly through the health department, when the labor department is the one having the authority and means to investigate and correct conditions deleterious to the health of employees. The physician is accustomed to report cases of sickness to the health department, so that adding occupational diseases to those previously notifiable is merely extending the scope of a practice with which he is already familiar. When the department of health receives these reports it must, however, immediately forward the information thus acquired to the labor department or be in a position to itself investigate and correct faulty industrial conditions. Most of the value of the notification of cases of occupational diseases is lost when the reports are intended merely for purposes of record. Under such conditions they serve mainly as statistical data to indicate what has happened in the past, data incomplete and containing many undiscovered errors because not investigated, data therefore likely to be untrue and undependable.

The State's Right to Require Reports.—The right of the state to require physicians to report cases of disease has been repeatedly declared by the

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courts. The state that issues to an individual a license to practise medicine certainly can stipulate under what conditions the practice shall be carried on. From time to time states have passed laws allowing physicians a fee for each case of notifiable disease reported. The payment of a fee gives the report the semblance of a business transaction, at least the physician is likely to receive that impression and to feel that if he does not want the fee, he need not make the report. A small fee is but little inducement to a busy practitioner, and it has been suggested that a large fee might be the cause of numerous erroneous notifications. The importance of these reports to the community is too great to allow of their being placed on a commercial basis. As well give chauffeurs a fee for each time they are inconvenienced or delayed by keeping within the speed limits.

The paying of a fee for morbidity reports has not been successful, for in reviewing the results being obtained in the notification of the communicable diseases it is found that on the whole the states which pay a fee for each report are getting poorer results and less complete reports than the states which pay no fee.

The Information Given in Reports.—The department whose duty it is to investigate reported cases of occupational diseases and cause such changes to be made as will prevent the occurrence of additional cases must have detailed information of each case. For the purpose of understanding the circumstances under which the illness was contracted it will want to know the nature of the existing illness and its time of onset, the age and sex of the patient, the nature of the industry in which he worked, the particular kind of work at which he was employed, the place where employed, the length of time he worked in his last position and the place or places where he had previously worked, the length of time he has worked at his present occupation and the kinds of work engaged in before. There is other information that will be desired for purposes of record and statistical analysis. Still other data having direct and important bearing will be brought out during the investigation of the case and the conditions under which it arose, all of which the investigator will record. The question which naturally arises is how much of this information should be contained in the primary report made by the physician and how much should be gathered by the health department or labor department, whichever is in charge of industrial sanitation; how much of the data can the attending physician furnish and how much should be expected of him. It would seem that the physician should not be asked to give information which can be obtained more satisfactorily or with greater accuracy in some other way, but that he should give that information which, because of his special knowledge of disease and as the physician in attendance on the patient, he alone can give. He might also properly give such other data as can be readily obtained by him and are necessary to enable the proper authority to investigate the industrial con-

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ditions responsible for the illness, including the nature of the disease, the name and address of the patient and the place where employed.

The Importance to the Physician of a Knowledge of Occupational Hazards.—In the proper treatment of the sick a clear understanding of the nature of the illness and usually of its cause and of the circumstances and conditions in the life of the patient which have contributed to or been responsible for the disease is necessary. To acquire this knowledge, information of the patient's occupation and its relation to the illness must be obtained. The practising physician needs, therefore, to be familiar with the industries of his community and the morbid effects which they are liable to produce in those engaged in them. Otherwise, he will frequently fail to make a proper diagnosis, will fail in treatment, and will be unable to give his patient the necessary advice to prevent recurrence of the illness.

The Relation of Occupational to Other Diseases.—Occupational diseases are diseases produced as a result of the nature of the patient's occupation or employment. They cannot be put into a class by themselves. Few diseases are produced only by occupation. Many morbid conditions may be produced as a result of the nature of the patient's employment or may be due to other causes. The diagnosis, therefore, does not usually show whether the morbid process should be classed as an occupational disease. However, a knowledge of the patient's occupation and its possibilities for harm to those engaged at it will suggest the likelihood of the illness being caused thereby.

Compressed-air illness and certain tics and palsies are almost invariably due to occupation. So was phosphorus poisoning in the United States before Congress placed a prohibitive tax on the manufacture of white phosphorus matches and in that way put an end to the industry. Anthrax is usually contracted as a result of the nature of the patient's employment.

Lead poisoning is also usually acquired as a result of the patient's work. There are considerably more than 100 different trades in which the workmen may get this metal into their bodies and be poisoned as a result. It is to be borne in mind that workers in lead may show no symptoms usually considered indicative of plumbism and yet be suffering from its effects. The existence of such a condition in a patient should be recognized by the physician, for its effect on ailments supposedly due directly to other causes may be considerable. Chronic nephritis and arteriosclerosis may be due to lead poisoning or to other causes.

Tuberculosis may very probably be produced as a result of lessened resistance to the disease due to injury done to lungs by the continued inhalation of various irritating dusts. Many occupations have associated with them ailments which occur among those engaged at them more frequently than among others. Some occupations by their insidious effects seem to shorten merely the life of the worker; others produce chronic disease which

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in time causes an incapacity for work and compels the laborer to abandon his trade.

State Laws Requiring the Notification of Occupational Diseases.—Laws requiring that physicians report to designated authorities cases of occupational disease among their patients are of comparatively recent origin. They represent a transition from the period when the control of unhealthful industries consisted solely in the enactment of laws requiring the installation of safeguards in certain industries, and the regulation of some of the conditions under which work was performed, to the period in which effective work is to be accomplished in the control of occupational diseases—work that will be effective because it will be done with a definite knowledge of the industries in which the health of the workers is being undermined, and not only of the industries but of the particular factories and establishments. The transition is from the indefinite generalized effort usual in the absence of satisfactory knowledge to the more direct and effective measures based upon definite information and a clear understanding. Each reported case gives notice of a factory or workshop where the conditions are such that the workmen are being deprived of health.

The legislation in the United States has been patterned to some extent after that of Great Britain. The bills passed by most of the state legislatures have been copies or modifications of a standard bill drafted by the American Association for Labor Legislation.

The first legislation in the United States requiring the notification of occupational diseases was enacted in 1911, when six states passed laws. These states were California, Connecticut, Illinois, Michigan, New York, and Wisconsin. The California law was approved April 21, 1911, and has the distinction of being the oldest of the laws. Two states, Maryland and New Jersey, passed laws in 1912, and seven states, Maine, Massachusetts, Minnesota, Missouri, New Hampshire, Ohio and Pennsylvania, followed in 1913, and Rhode Island in 1915.

The laws of Illinois, Missouri, New Jersey, Ohio and Pennsylvania require that employers in industries in which the workers come into such contact with certain harmful processes or materials that injury may be caused to their health shall have the employees examined by a physician at least once a month to ascertain whether any occupational disease (in New Jersey lead poisoning only) exists. Of this type of law that of Missouri is the most comprehensive and contains the following provisions:

Provisions of Missouri Law.—Employees engaged in manufacture in which antimony, arsenic, brass, copper, lead, mercury, phosphorus, zinc, their alloys or salts, or any poisonous chemicals, minerals, acids, fumes, vapors, gases or other substances are generated, used or handled by employees in harmful quantities, or under harmful conditions, are required at least once a month to cause all employees coming into direct contact with the poisonous agencies to be examined by a physician to ascertain whether there

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exists in the employees any disease due or incident to the character of the work in which the employees are engaged. The physicians making these examinations are to make within 24 hours a report to the state board of health in triplicate upon blanks furnished by said board. If disease incident to occupation is found, the report is to state the name, address and business of the employer, the nature of the disease, and its probable extent and duration, the name of the employee and his last place and length of employment.

Upon receipt of these reports the secretary of the state board of health is to send one copy to the state factory inspector and one copy to the superintendent of the factory in which the employee is supposed to have contracted his ailment.

The enforcement of the law is made the duty of the state factory inspector.

Failure on the part of a physician to make the required reports is made a misdemeanor punishable by a fine of not less than $50. (Act approved March 27, 1913; effective June 23, 1913.)

Provisions of Other State Laws.—The Massachusetts law merely provides that the state board of labor and the industrial accident board, sitting jointly, shall make regulations for the prevention of occupational diseases. This joint board is given authority to require physicians to report all cases in which the patient is suffering from any ailment or disease contracted as a result of the nature, circumstances or conditions of employment.

The other state laws provide that medical practitioners shall report to a designated state authority all cases in their practices of certain diseases, usually occupational in origin. These diseases include poisoning by lead, phosphorus, arsenic, mercury, wood alcohol, or brass or their compounds, compressed-air illness and anthrax. In some of the states the above-named diseases are to be reported only when they have been contracted as a result of the nature of the patient's employment. This includes practically all cases excepting those of intentional or accidental poisoning by mercury, arsenic and wood alcohol. In some of the states all diseases due to occupation are to be reported. The New Hampshire law illustrates this type and makes the following provisions:

Provisions of New Hampshire Law.—Physicians are to report all cases among their patients believed to be suffering from poisoning from lead, phosphorus, arsenic, brass, wood alcohol, or mercury or their compounds, or from anthrax or compressed-air illness, or from any other ailment contracted as a result of the nature of the patient's employment. These reports are to be made to the state board of health within 48 hours and are to give the name, address and occupation of the patient, the name, address and business of the employer, the nature of the disease and such other information as may be required by the state board of health.

The state board of health is to prepare and issue blank forms on which the reports are to be made by physicians, and is to transmit copies of reports received to the Commissioner of Labor.

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Violations of the law on the part of physicians are made punishable by a fine of $5 for each offense. (Act approved May 7, 1913; effective July 1, 1913.)

Analytical Summary of State Laws.—The occupational diseases required by the state laws to be reported are anthrax, compressed-air illness and certain industrial poisonings; poisoning from lead or its compounds is mentioned in 14 of the laws; poisoning from phosphorus, arsenic or mercury, or their compounds, and compressed-air illness are specified in 12 of the acts; poisoning from brass or wood alcohol is included in 5, and in 11 anthrax is named. In 7 of the laws it is stated that all occupational diseases shall be reported.

The information to be given in the physicians' reports differs to some extent. In 14 of the states the patient's name and address are to be given, in 8 his occupation and place of employment, in 2 the duration of employment and of the disease, in 13 the nature of the disease, in 5 the name of the employer, and in 4 his address and business.

In 11 of the states the physicians are to send their reports to the state department of health or board of health, in 3 to the state commission of labor or labor bureau, and in 1 state to both the labor and health departments.

In 8 of the states in which the reports are made to the state board of health, the health department forwards the reports or transcripts thereof to the proper labor office. In 1 state, Maryland, the department of health transmits the data to the state bureau of statistics and information.

In 4 states the physicians are to make their reports at once, in 1 within 24 hours, in 5 within 48 hours, in 1 within 10 days, and in 1 within 30 days.

Penalties for failure on the part of the physician to report have been provided in 11 states. They include both fines and imprisonment. The fines vary from not over $5 in Maryland to one of not less than $50 in Missouri. In Minnesota the penalty is a fine of not over $10 or imprisonment for not over 10 days.

In 4 states it is made the duty of the state health department to enforce the law, in 6 of the state labor department.

In 2 states, California and Connecticut, a fee of 50 cts. is paid to physicians for each report made.

Enforcement of Notification Laws.—The comparatively small number of cases of occupational diseases as yet reported in those states having laws requiring notification is due in part to the failure on the part of physicians to recognize the true nature of such cases. Another factor is undoubtedly that the requirement of notification is comparatively new, being in no instance much over 4 years old, and that physicians have not as yet become accustomed to report, although in most of the states the physicians have been circularized once or more for the purpose of making known to them their duties as specified in the notification laws. There can be little, if any, question that considerate but nevertheless watchful enforcement would materially increase the number of reports. Flagrant violations of the law certainly should be prose-

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cuted, and particularly because of the value this course would have in impressing all concerned with the need of complying with the statutes. Those whose duty it is to enforce these recently enacted laws for the notification of occupational diseases have a heavy responsibility, for much always depends upon the manner of enforcement of a new law during the period immediately following its enactment. It is much easier to enforce a law from the beginning than to bring back to life one that has been a dead letter for a time. The future of the notification of cases of industrial diseases will be influenced to a marked degree by the intelligence and earnestness of those who now have the responsibility of enforcing these laws. The enforcement of laws is more important than their enactment.

Those who will be benefited most by the reporting of occupational diseases and by the improvement in the sanitary conditions in trades and industries which will follow are naturally the laborers, the workmen and the workwomen. These should take a keen interest in having every case of occupational disease reported as required by law. The matter is one also of interest to labor unions, to fraternal organizations having sick or death benefit features, and to the insurance companies devoted largely, or in part, to industrial insurance, all of which can be of material assistance to secure the enforcement of the notification laws.

Blank Forms in Use.—The blank forms used by the several states differ to some extent, depending upon the requirements of the law regarding the notification of cases of occupational diseases. Several of the states, among which may be mentioned New Jersey, New York, New Hampshire, and Maryland, use the same form of blank, a copy of which is here reproduced.

Mortality Records of Occupational Diseases.—The standard blank form approved by the United States Bureau of the Census for use in the making of death certificates contains a space for a statement of the occupation of the deceased. With this information of the nature of the occupation it is possible to classify the deaths according to the kind of work in which the decedents were engaged. To be able to do this is highly desirable, for although it does not show the origin of the diseases causing death, that is, whether they were due to occupation, it nevertheless makes it possible to know whether those engaged in any particular work have on the average a shorter duration of life, or die in greater numbers of any one disease, or are killed in greater numbers by accidents than is true of people in general.

The statements of occupations made in death certificates are in no wise a substitute for the reporting by physicians of cases of occupational disease, nor does the notification of disease serve as a substitute for the statement of occupation made in the death certificates. They furnish entirely different information and serve quite different purposes. The notification of cases of disease due to occupation shows when and where occupational diseases are occurring at a time when it is usually possible for an investigation to be made to ascertain whether the case is truly due to occupation, and to study

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WRITE PLAINLY WITH INK—THIS IS A PERMANENT RECORD

N. B.—Every item of information should be carefully supplied. The exact statement of OCCUPATION is very important. Physicians should state DIAGNOSIS in plain terms. See instructions on back of certificate.

CERTIFICATE OF INDUSTRIAL DISEASE

NAME OF PATIENT..

ADDRESS: Street and No......................**City or Village**......................

PERSONAL AND STATISTICAL PARTICULARS

Sex	Age	Color	Country of birth

Single, married, widowed or divorced (*write* the word)

Occupation

(a) **Present** trade, profession or work............

....

Particular kind of work in such trade, etc.......

....

Date of entering present occupation............

Employer's name..............................

Address......................................

Business (kind of goods made or work done)....

....

(b) **Previous** occupations:

Name of occupation	Entered (year	Left (year)
....	....	....
....	....	....
....	....	....

Previous illnesses, if any, due to occupation.

Disease or illness	Year
....	....
....	....
....	....

MEDICAL CERTIFICATE OF DISEASE

Diagnosis of present illness.............

Chief symptoms and conditions.........

....

....

....

Date first symptoms appeared..........

Complicating diseases (such as alcoholism, syphilis, tuberculosis, etc.)

....

....

....

....

....

Additional facts..........................

....

....

....

Date of diagnosis.............., 191...

(Signed)........................ M. D.

............, 191... (Address).........

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and remedy the conditions which made it possible for the disease to have been so contracted. The statements of occupation in death certificates show the duration of life of those engaged at various kinds of work and the terminal diseases and accidental injuries of which they die.

The value and accuracy of these data in death certificates depend almost entirely upon the care and attention given by the physician in stating the cause of death and by members of the decedent's family in stating the exact nature of the occupation of the decedent. It is impossible to compile statistically the occupations and causes of death if erroneously given and arrive at results which are dependable and show conditions as they actually are. A death certificate properly filled out should show not only the industry in which the decedent was engaged, but also the particular kind of work at which he was employed. For example, 50 years ago a statement that a man was a printer described his occupation in a fairly satisfactory manner, for in most shops the workmen practically all did the same kinds of work. But this is not sufficient at the present time with the development of large shops and the differentiation of labor. A printer may be a pressman or he may be a compositor. A compositor, on the other hand, may be a hand typesetter or a typesetting machine operator. But the typesetting machines most commonly used are of two kinds: the linotype, in which the molten type metal is cast in the same machine at which the operator works, and the monotype, in which the keyboard is entirely detached from the molten metal which may be cast in a different room or even in a different building. The monotype keyboard operator may in no wise be exposed to the dust or fumes of lead, while the hand compositor and the linotype operator are both subject to such exposure. For these reasons the exact nature of the work performed is essential to a proper understanding of the occupation. The same need for describing the particular labor performed is equally necessary in other trades and occupations if the derived statistics are to be of value.

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CHAPTER III

THE PROTECTION AND PROMOTION OF THE HEALTH OF WOMEN WAGE EARNERS

BY IRENE OSGOOD ANDREWS, New York City, N. Y.

Because of the physical differences between men and women, working women are exposed to health dangers which do not so seriously threaten men. With a physique slighter and less strong, woman has not the same power to resist conditions which insidiously break down health and which predispose to more serious disturbances. She has, therefore, been given by most civilized countries special legislative protection.

In sharp contrast with the leisurely, easy-going methods found in the early stages of factory production, practically all countries have seen a new and threatening strain created in industry by the introduction of machinery followed by constant speeding up, by an ever increasing number of machines to be handled or attended by one person, and by the requirement of a constantly enlarged output. The complexity of modern industry and machinery, in marked distinction from the simplicity of the earlier forms, has resulted in a division of labor which has greatly increased the speed and monotony of work and tends to bring on a fatigued condition. Authorities have shown that the wastes of the body, which under normal conditions of work and exercise would be thrown off, under conditions of overwork and exhaustion are allowed to accumulate in the blood and the worker is literally poisoned by these waste products and predisposed to specific ailments as well. This condition also increases the danger of contracting those definite occupational diseases which threaten many industrial workers.

The movement toward protective legislation has been greatly stimulated by the significant increase during the last 30 years in the number of females industrially employed in this country. In 1880 this number reached over 2,500,000; in 1900 the number rose to over 5,250,000 and in 1910 the number had grown to 8,075,772, or 21 per cent. of the entire gainfully employed population. Upon the basis of the census we find that the number of female breadwinners is increasing faster than the number of male breadwinners, and also much faster than the female population.

Females 16 years of age and over represent about 25 per cent. of the total number employed over that age and are distributed as follows:

Agriculture	1,807,050
Domestic and personal service	2,620,857
Manufacturing and mechanical	1,772,095
Mining	1,094
Trade and transportation	1,502,352
Professional	673,418

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While the proportion of women to men in the manufacturing and mechanical industries, which most concern us here, has remained since 1899 the same—that is, 19.5 per cent.—the total number of women has increased, and in certain of the larger industries it has increased faster than the number of men. Among these industries are boots and shoes, canning and preserving, confectionery, printing and publishing, silk and silk goods, tobacco, and woolen and worsted goods. Among those industries where the number of women is apparently increasing, but not at so rapid a rate as the men, are cotton goods, men's and women's clothing, hoisery and knit goods, and millinery and lace goods. A smaller industry in which the number of women is increasing not only very rapidly but also much more rapidly than the number of men is the manufacture of electrical machinery, apparatus and supplies. Here the number of women over 16 increased from 6,956 in 1899 to 19,831 in 1909—an increase of 185 per cent.; the number of men in the meantime increased only 93 per cent. Men still form 76 per cent. of the total number employed but this represents a decrease from the 82 per cent. employed in 1899, while the proportion of women to the total number employed has increased from 16 per cent. to 22 per cent.

The necessity for control over women's work is now generally accepted in this country, both by legislators and by the courts. This acceptance has been secured because of the emphasis which has been placed upon the need of protecting the health of women.[1] As Justice Brewer said (Miller vs. Oregon, 208 U. S. 412): "That woman's physical structure and the performance of maternal functions place her at a disadvantage in the struggle for subsistence is obvious. This is especially true when the burdens of motherhood are upon her. Even when they are not, by abundant testimony of the medical fraternity, continuance for a long time on her feet at work, repeating this from day to day, tends to injurious effects upon the body, and as healthy mothers are essential to vigorous offspring, the physical well-being of woman becomes an object of public interest and care in order to preserve the strength and vigor of the race."

In regard to legislation, however, concerning those occupations in which the employment may directly produce disease, we find ourselves far behind European countries. Practically no thoroughgoing, extensive investigations of the specific occupational diseases among women workers have been made in this country. Indeed it has been only within the last few years that the subject here has been widely discussed at all. It is mainly because of this lack of public information and interest that we find special legislation on this subject in America so meager.

Control over woman's work is secured by three main methods. The first is prohibition, by which women are entirely excluded from certain occupations which present unusual danger, such as the use of poisons, excessive dust or very high temperatures. The second method is regulation, by which conditions in work places must conform to certain specified

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minimum standards before women may be employed therein. Regulations relate usually to air conditions, sanitary conditions, temperature, or hours of labor. The third method is insurance, by which more adequate protection is afforded workers through the action of the employer who for economic reasons desires to reduce costs due to compulsory compensation for industrial accidents or sickness.

Before entering upon the details of specific legislative enactments attention should be given to the difference which exists between the European method of establishing protective regulations and that employed in America. Here, for legislative purposes, we as a rule group many industries together, as manufacturing, mercantile or transportation. But in Europe, for the more important legislation, each particular industry is studied by itself and rules are provided to fit the peculiar dangers of that industry. In addition, if any occupation or process within the industry holds special dangers, special rules are made to apply for that occupation or process alone. Thus in France women and children are excluded from a list of over 100 occupations unless certain regulations are complied with, and they are entirely excluded from an additional list of over 50 dangerous trades.

The method of administration in Europe also differs considerably from that in America. In foreign countries a general law is usually accompanied by the authority to make, through administrative orders, rules and regulations for each separate industry or occupation as the needs of the industry or occupation may demand, including also the power to make limited exceptions. This applies to the limitation of hours of work as well as to the regulation of physical conditions. Until recent years in America all protection was secured through legislative enactment alone, with considerable discretionary power in the hands of the inspection officials. Within very recent years several American states, notably California, New York, Oregon, Pennsylvania and Wisconsin, have enacted labor legislation based upon the method used in Europe, and used in America for many years by public service commissions and by boards of health.

Having in mind, therefore, the European method of enacting protective legislation, one is not surprised at finding the factory acts of those countries a mass of detail frequently applying only to one industry, one occupation or one process, containing many exceptions and in most cases providing for special orders by the administrative authorities.

PROHIBITED EMPLOYMENTS

In taking up the first method of control over woman's work we find that practically all civilized countries have recognized that certain employments, because of unusual physical or moral dangers, should be closed to women, and they have given legal sanction to such exclusion. In America most of these prohibitive laws relate to mines and saloons. Women

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are forbidden to work in mines in most of the mining states and in saloons, unless they are members of the family, in about 16 states. The employment of women is forbidden also in a few states in cleaning moving machines and in the use of emery, polishing or buffing wheels where articles of iridium or of the baser metals are manufactured.

Effective legislation of this character, however, presupposes careful intensive investigation and very few such investigations have been made in this country; they are particularly lacking for the more dangerous occupations such as those involving the use of poisons or the presence of harmful dusts or gases. We find ourselves, therefore, far behind European countries in this particular. Take, for example, the one instance of the use of lead. The harmful effects of contact with this poisonous substance, and particularly the peculiar susceptibility of women, have long been known. In America[2] women employed in lead processes in potteries, for example, are engaged usually in dipping the ware in the lead glaze, removing the excess glaze, decorating with lead colors and cleaning or sweeping floors, boards or tables where lead dust is present. Investigations have shown that among women employed in potteries in America the ratio of cases of lead poisoning is 1 to 7, while in Great Britain the ratio is 1 to 64. Yet in only six states are there any protective regulations and in no state are women excluded from work involving the use of lead. Abroad, however, this prohibition is placed with more or less thoroughness upon women and children in many countries, including Argentina, England, Germany, France, Italy and Holland.

Among the other occupations involving the use of poisonous substances and forbidden to women in various countries are the making of electric accumulators, the manufacture of paints, varnishes and colors, brass casting, zinc or lead smelting, certain parts of glass manufacture, the manufacture of explosives, the curing of skins or fur of rabbits, and many other occupations involving the use of injurious chemicals. Most foreign countries also prohibit the underground work of women in mines. In France a list of over 100 occupations is entirely forbidden to women and to children under 18, including such occupations as lace bleaching with white lead, curing the skins or fur of rabbits, sharpening or polishing metals, coating mirrors with quicksilver, and the cutting or tearing of rags. Twelve more employments are forbidden entirely to children under 18, and an additional list of occupations in which these persons may not engage, unless certain harmful conditions are removed, includes establishments for bleaching cloth, straw or paper, rubber works where fumes of sulphide of carbon are given off, and print cloth factories, dye works, and metal-lacquering establishments where poisonous substances are used. Spain has a well-classified list of over 100 occupations forbidden to children under 16 and women under age. Similar lists, while not identical, are found in the more important countries. In most of these countries the administrative authorities are given power to extend

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the lists of prohibited employments at any time that investigation reveals the necessity for exclusion.

REGULATED EMPLOYMENTS

It is seen that the majority of the occupations which are forbidden to women in Europe involve the presence of dusts, fumes, vapors, gases, or substances of a poisonous or well-defined harmful character. There exists, in addition, a large group of occupations where injurious substances or conditions are present but where the ill effects upon health are less definite. This second and much larger group comprises those occupations which expose workers to such harmful conditions as extremes of heat or cold, undue humidity, impure air, insufficient or poorly distributed lighting, or excessive speed or strain. While these conditions produce a marked effect upon the health of women, predisposing them to such diseases as pneumonia, tuberculosis, functional and nervous disorders, eye injuries or orthopedic derangements, yet it is difficult to designate sicknesses of this character as specifically occupational in origin, since conditions outside the work place may be a factor in producing the illness.

The control of conditions in occupations of this class is usually effected by means of regulations either directly specified in the laws or issued by the administrative authorities as rules or orders. In America legislation of this class is extremely general in character, is loosely drawn, and seldom amounts to more than a mere declaration of principles. Such laws apply usually to all industries and relate to adequate ventilation, proper toilet facilities, cleanliness, lighting, overcrowding, and seats for females. These measures in most cases apply to both sexes alike and depend for their enforcement almost entirely upon the spirit and intelligence of the administrative authorities. They are to be found in all of the industrial states of America.

In a very few cases more specific provisions are found, as in New York where females may not be employed in connection with core making in any brass, iron or steel foundry unless the room where cores are baked is separated from the making process by partitions extending from the floor to the ceiling and so constructed as to prevent the passage of gases and fumes into the room where the women are working. The State Board of Labor and Industries in Massachusetts, also, may make regulations for the control of work in core rooms, and a few states provide for additional cleanliness and toilet facilities where women are employed.

The lifting of heavy weights is regulated in only one state, Massachusetts, where pulleys or castors must be provided for moving packages or receptacles 75 lb. or more in weight which are handled by females; no classification as to age is made and the act applies only to manufacturing or mechanical establishments.

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In a few additional states specific regulations have been adopted which apply to men and women alike, as in Massachusetts where the relation of humidity to temperature in textile mills is regulated, in New Jersey, Michigan and Illinois where specific rules exist for dust removal, and in Wisconsin where very extensive regulations have been promulgated; yet the instances cited above practically exhaust the special protective regulations for women in this country.

In contrast to these prevailing vague general provisions applying to both sexes alike, we find in Europe detailed regulations as to temperature, humidity, lighting, and the lifting of weights, applying to women and minors alone. In regard to the important factor of temperature in workrooms, we find specific regulations in many countries. In Holland, for instance, women and young persons under 16 years of age may not be exposed to a temperature of 57⅗°F. if the outside temperature is below 52⅕°F. in the shade; if the temperature is 52⅕°F. in the shade or higher, the inside temperature may not exceed this by more than 5⅖°F. They may not be employed where the temperature is continuously from 45° to 57⅗°F. unless the measures prescribed by the district inspector of labor for lowering the temperature have been adopted; neither may they be employed in any place which is not heated to 18°F. or any higher temperature which the district inspector may require, particularly if the employees are to do work which requires little physical exertion. In addition, special requirements as to temperature and humidity are placed upon special processes; in spinning or weaving establishments, for instance, if the atmosphere is artificially dampened, a wet-bulb thermometer must be provided and the temperature may not exceed 45°F. unless the outside temperature exceeds 57⅗°F. in the shade, in which case the inside temperature may vary in the same proportion as the outside temperature. Experts in this country might disagree upon the exact degree of temperature to be allowed, but as yet we have not even taken steps for regulations of any kind.

In Holland, also, we find special provisions in regard to proper lighting. Women and young persons under 17 may not be employed in places which are insufficiently lighted or where artificial light is required between 9 A.M. and 3 P.M. unless the nature of the work done makes it impossible to admit light, in which case the hours of work may be regulated by the administrative authorities and every such person must be given a certain number of hours each day in the sunlight. In addition, the illumination must equal at least 30 Hefner units at a distance of 1 meter in the following occupations: embroidering, weaving damask, silk or cotton colored materials, the treatment of diamonds or other precious stones, glass-cutting, engraving or wood-cutting, instrument making, lace working, bead threading, typesetting, machine knitting, gold and silversmith's work, making of hair goods, sewing, quilting (except leather quilting), drawing (designing), and the manufacture or repair of clockwork. In all other kinds of work the illumination must equal at

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least 20 Hefner units at a distance of 1 meter. Workers must also be protected from glaring light.

While we found only one American state, Massachusetts, providing protection for women and young persons in the matter of lifting heavy weights, such regulations are frequent in foreign countries. In France weights to be handled are regulated according to age and to the method of moving; women over 18 may not carry any weight over 25 kg. nor move, on two-wheeled handcarts, more than 130 kg. (including weight of handcart). In Argentina this regulation is confined to persons of both sexes under 20 and weights to be lifted or moved are carefully adjusted to the age of the worker.

Special provisions are also frequently made in European countries for employed mothers who are nursing their children. Thus in Italy in establishments where at least 50 women are employed a special room hygienically kept must always be available for the nursing of infants, and women using this room must be allowed at least ½-hour in addition to the regular rest periods; if women nurse their children off the premises they must be allowed a full hour in addition to the regular rest periods. In France teachers and females on the staff of the Department of Posts, Telegraphs, and Telephones are granted, at the time of childbirth, a leave of absence for 2 months together with full treatment.

American Conditions Need Protective Legislation.—While it is true, as already stated, that specific health investigations have been so rare in this country that competent data do not exist for the formulation of special regulations, yet sufficient data are at hand to indicate many industries which threaten the health of women and children. Among such dangerous callings may be mentioned the manufacture of cordage, twine and jute goods in which over 17,000 women and children are employed. In this industry many women are frequently employed without adequate protection from the steam, heat and wet of the spinning rooms, where the spray and drippings frequently wet their clothing up to the waist and where pools of water, gathered on the floors, often compel them to go barefoot. Among such workers, colds, rheumatism, bronchitis, and menstrual troubles are prevalent. One of the countries which have regulated conditions of work in these industries is England where the rules require adequate dressing rooms for holding dry clothes and changing after working hours; wet room floors made of material impervious to moisture, which will maintain the level, and will not crack; splash boards for the protection of the workers; and trays or groovings in the floor under the frames to catch the drippings and to drain off the water. Adequate ventilating devices to draw off the steam are also often required. In France, also, women and children under 18 may not be employed at the wet spinning of flax unless the overflow of water is properly removed.

In certain other processes of this same industry a large quantity of harmful dust is present, causing physical injury to the workers. Physicians

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practicing among operatives in the dusty processes testify that chronic bronchitis, asthma and catarrh are unduly prevalent. Other physicians have found a certain skin disease which they attribute to the presence of irritating dusts or harsh oils. In France children under 18 may not be employed in the stripping of flax, hemp, or jute where dust is freely given off.

The difference between the method of legal regulation as adopted in Europe and that adopted in most American states is well illustrated by a comparison of the English law governing dusty work of this kind with that of Massachusetts where a large number of such establishments exist. The Massachusetts law merely authorizes the inspector to require the use of a fan or some mechanical means of ventilation if he thinks injurious dusts are present and are inhaled in a harmful degree, and if such device will not entail an unreasonable expense to the employer.

The English law requires that in every room in which roughing, sorting, hackling, preparing or carding of flax or tow is carried on, efficient exhaust and inlet ventilation shall be provided to draw away the dust from the workers, at or as near as possible to the point at which it is generated. The sectional area of the exhaust openings near to each set of hackles must be not less than 120 sq. in., and the arrangement must be such as to secure a velocity of the air passing through each exhaust opening, and measured at that point, which at any time is not less than 450 lin. ft. per minute. The air inlet must be so managed that no direct draft falls upon the persons employed, and the arrangement of inlet ventilation must, as far as practicable, be such that the temperature of the incoming air shall not be less than 60°F.

Investigations of the federal Bureau of Labor Statistics have also given data on unhealthful occupations. An illustration of such conditions is found in the glass industry which employes nearly 10,000 women over 16 years of age in certain departments.

In the making of incandescent lamps, we find examples of extreme speed and minuteness of work. In the tungsten department, for instance, in one operation "the gossamer-like tungsten filament, which can hardly be seen by the untrained eye, must be inserted in a tiny hole punched in the end of a copper wire no thicker than the finest needle, where it is kept in place by pinching the sides of the wire together. This operation is repeated a thousand times a day at the rate of about three every 2 minutes. Almost everyone has done at some time equally delicate work, so that the effects which operations of this nature must have on the eyes and nerves can best be appreciated by conceiving of the repetition of such work through a 10-hour day at a rate approximating that cited above." It is pointed out that without any extensive reduction in the cost of materials used, a great reduction in the price of electric lamps has been effected largely because of the subdivision of processes, increased speed and the utilization of female labor.

In the glass industry proper, the chief processes employing large numbers of women and involving danger to health are those in finishing and decorating

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which consist mainly of polishing, sand blasting, acid etching, cutting the glass, and painting and enameling. In sand blasting, if the apparatus is not properly covered, the finely powdered sand escapes through the top of the machine in a forcible blast, passes freely into the room and fills the air while the operator receives in the face the full force of the dust stream. This dust is extremely fine and hangs suspended in the air for a considerable time. The federal investigation revealed the fact that nearly all the women employed at this work complained that the dust caused a painful irritation of their eyes and throat. The inhalation of dust together with the necessity for constant standing make the work of sand blasting particularly harmful to women. The process of acid etching, while employing few women, exposes these few workers to the fumes of hydrofluoric acid which causes violent irritation of the eyelids and conjunctiva, cold in the head, bronchial catarrh with spasmodic coughing, sores on the gums and mucous membrane of the mouth, and ulcers, corrosion and blisters. The investigators found that in one factory where women were employed at this work they frequently had trouble with their eyes, and several former employees had been so badly injured by the fumes that they were compelled to stop work entirely.

In painting and enameling glassware, also, danger exists from the fine enameling compounds which are dusted or sprayed on the glass and which usually contain lead and arsenic. This fine dust is inhaled by the operatives. Women engaged in the occupation of silvering mirrors often work in rooms where the temperature reaches 104°F., this temperature being considered necessary for the best results. Most of this work is done while standing, and by women over 20 years of age.

Beyond the most general laws, no American state attempts to regulate such work, while in European countries it is frequently controlled by special rules. In Italy women and children may not be employed at all in gilding and silver plating, and those under 21 may not be employed in grinding and polishing glass, nor in making it opaque or corroding it with hydrofluoric acid or sand jets. In Holland women and children may not be employed where excessive dust is present. In France, women and children under 18 may not be employed at the dry-polishing of glass, nor in any process where dust is given off freely, nor where poisonous materials are used. In England women and children may not take their meals in any part of a flint glass factory where grinding, polishing or cutting is carried on.

The federal investigation indicated many other industries which exposed women to unnecessary harmful conditions and which were not controlled by legislation such as we find in European countries. Among these may be mentioned the making of brassware, particularly in work in the core rooms and around the ovens which expose the women to high temperature and injurious fumes; in nickel plating where injurious and often poisonous fumes are present; in the manufacture of firearms and ammunition where mercury

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in the form of a fulminate is used; in making hardware where the lacquer involves the use of lead and irritating volatile solvents; in the making of saws and files and polishing wooden handles where lime, metallic and other irritating dusts are present; in the rubber industry where poisonous fumes exist; in artificial flower and calico cloth making where aniline dyes are used; and in pearl button making where ammonia, pumice stone and hydrochloric acid are present.

Restrictions upon Workpeople.—Control of woman's work by regulation is also secured by a second method—that of restrictions upon the workpeople themselves. The chief restrictions at present fall into four main groups: limitations upon the duration of the work period, restrictions in regard to eating places, prohibition of work preceding and following childbirth, and the medical examination of employees.

Limitations upon the Duration of the Work Period.—After all possible protection has been provided through prohibiting work in dangerous places and by regulating the physical conditions under which women may be employed, experience has shown that it is still necessary to limit the number of hours which a woman may be employed in a day or a week. Unless this is done, the fatigue resulting from excessive hours of work will in time undermine a woman's health, even under the most sanitary conditions. In American states this limitation has practically always taken the form of the same hour rate for all industries to which the law applies. In Europe such inclusive limitations are often supplemented by exceptions both above and below the limits of the general law.

Day and Week Limitations.—On the question of hour limitations for daytime work many American states have gone farther than the European countries. A few western states and the District of Columbia permit only 8 hours a day, while nearly a dozen more limit hours to 9 a day. A larger group of over a dozen states permit 10 or 11 hours a day, but in many cases the total number of hours per week must not exceed 54 or 56. In actual operation this frequently means that Saturday afternoon becomes a half-holiday, particularly in the summer time. Still another group allows 10 hours a day and 60 a week, while six or eight states place a 9- or 10-hour limit on day work but place no limit on the hours per week; this may permit full time work for 7 days a week. While only a few states restrict work to 6 days a week the limitation by both daily and weekly periods has a tendency to eliminate 7-day work. It is, however, well known that violations of the laws frequently occur both as to hours per day, hours per week and days per week. At present about half a dozen American states still have no legislation restricting the hours of labor for women workers.

In most of these state laws exceptions exist which permit overtime for certain periods, in certain industries or under specified conditions. Other laws, such as those in North and South Dakota and Oklahoma are so worded as to permit enforcement only against those employers who *compel* work for

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longer than the stated period, and are therefore practically unenforceable. American laws apply as a rule to most industries where women are employed.

European legislation respecting the hours of labor for women presents a mass of detail similar to that found in legislation for the control of work conditions. While general laws exist in most countries, hours in many industries or occupations are governed by special regulations, which may grant a work period either longer or shorter than that stated in the general law. In many European countries, as in several American states, the canning and preserving industries, for example, are permitted a longer work day than that allowed in the general law. On the other hand, many special rules are found which reduce the work period below that stated in the general law. Thus in the canton of Berne no girl under the age of 17 years may be employed at a treadle machine for more than 3 consecutive hours a day. Women may be employed in the work of making paving stones only by special permission, and then the hours are limited to 6 per day. Frequently also European countries fix in the law the time of the day within which working hours must fall. Thus in England in textile factories employment must fall between 6 A.M. and 6 P.M. or between 7 A.M. and 7 P.M.; employment on Saturdays must end not later than 1 P.M.

Only one American state, Oregon, through its Industrial Welfare Commission, had in 1915 established for any occupation a shorter work day for women than that stated in the general law. A few employers have voluntarily or because of trade-union pressure reduced hours below the legal maximum set by the state.

Night Work.—The leading nations of the world have recognized the physical and moral dangers of night work for women and have enacted laws prohibiting such work for given periods. Scientific investigations everywhere have shown that the work of women at night is exceptionally injurious. Insufficient, broken and irregular sleep, lack of sunlight, irregular meal times, disarrangement of the normal customs of life, injury to eyesight, increased chance of accidents—all of these factors combine to lower vitality, to weaken the power of disease resistance, to produce impoverished blood and anæmia, to weaken the female reproductive functions and generally to increase morbidity and mortality. In addition a legal limit placed upon night work is a great aid in enforcing the laws limiting the hours of work per day.

But American states have been slow to remedy this serious evil. The movement here received a decided setback when in 1907 the New York Court of Appeals declared unconstitutional a law forbidding the work of women over 21 years of age in factories between 9 P.M. and 6 A.M. But in 1913 New York reenacted her earlier law, placing the closing hour at 10 P.M. and the Court of Appeals in 1915 reversed its earlier decision. The case is now pending in the United States Supreme Court, where it is hoped that a more enlightened public opinion will aid in upholding this much-needed protective

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legislation. Since 1890 in Massachusetts women have not been permitted to work in manufacturing establishments between 10 P.M. and 6 A.M., nor in textile factories between 6 P.M. and 6 A.M. since 1907. Similar laws prohibiting night work existed in 1915 in Arkansas, Connecticut, Indiana, Nebraska, Pennsylvania and South Carolina, and in Oregon, and in Washington for children only, by rulings of the Industrial Welfare Commission.

On the question of protection from night work European countries have greatly surpassed the American states. There 14 of the leading nations have entirely prohibited by international treaty the work of women between certain periods at night. This treaty is the result of a conference called by the International Association for Labor Legislation in Berne, Switzerland, in 1906. To this conference delegates were sent by the governments of Austria, Belgium, Denmark, France, Germany, Great Britain, Holland, Hungary, Italy, Luxemburg, Portugal, Spain, Sweden and Switzerland, and an international convention forbidding night work for women was signed by all of the 14 countries. By 1912 all of the countries except Denmark had enacted legislation embodying the provisions of the convention and had ratified the treaty. By the terms of this treaty, which applies to women over 18 years of age, the 14 countries bind themselves to allow to women at least 11 consecutive hours of rest at night and to permit no night work between 10 P.M. and 5 A.M.

In addition many of the smaller states and dependencies have indicated their adherence to the provisions of the treaty, while several of the signatory states have gone even further than the treaty requirements. France, Belgium, and Spain, for instance, have forbidden night work between 9 P.M. and 5 A.M., Austria has forbidden work at night between 8 P.M and 5 A.M., Germany between 8 P.M. and 6 A.M., and Holland between 7 P.M. and 6 A.M. Even in India the night work of women is forbidden in factories between 7 P.M. and 5:30 A.M.; in Argentina, between 9 P.M. and 6 A.M. Most of these countries permit exceptions under certain conditions, particularly where the nature of the materials used is such that a delay in handling would cause great financial loss. In such cases, however, very strict limitations are imposed, as, for example, in Holland where large numbers of women are employed in skewering herrings, a seasonal trade. Here employment after 10 P.M. is permitted for a limited number of hours and for a limited time. In addition, breaks in the work must be allowed every 4 hours, and if any break be less than ½ hour it must be counted as part of the period of employment; moreover, hot coffee or some other hot non-alcoholic beverage must be supplied to the night workers free of charge, extra wages must be paid for overtime work, and no woman may be employed after 10 P.M. without first securing a medical certificate stating that her health will not be injured by such work. Elaborate provisions for enforcement also exist.

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Rest Periods.—With the exception of requiring from ½ to 1 hour for the noon meal each day (or after 6 or 6½ hours' work), practically no legal attention has as yet been given in this country to the question of rest periods. With the increasing speed and complexity of modern industry, entailing great nervous strain, short intervals of rest during the morning and afternoon periods would mean much both to the physical welfare and to the efficiency of the worker. In the telephone service, for example, the nervous strain is particularly severe. A Canadian royal commission, reporting in 1907 on a dispute involving the Bell Telephone Company, recommended that the total work period be not longer than 6 hours spread over a period of from 8 to 8¾ hours; this would allow for at least three rest periods of from ½ to 1 hour's duration. A few American firms have voluntarily adopted the plan of giving their employees a 15-minute rest in the morning and in the afternoon and during this time thoroughly airing the workrooms. This principle is enforced by law in several European countries. Thus in Italy, women and children under 15 years of age who work from 8 to 11 hours per day must be allowed at least 1½ hours' rest each day; if work lasts longer than 11 hours, 2 hours' rest must be given. In Belgium, women in fruit preserving must be allowed at least 15 minutes' rest in every 5-hour period of work in addition to the noon rest. In the chocolate and confectionery industry, when work lasts between 9 and 10 hours a second rest period of at least 15 minutes must be allowed, and if the working hours exceed 10, a third rest period of 15 minutes must be granted.

Annual rest periods, or vacations with pay, have been voluntarily granted by a few firms in this country, but such a provision for private employments has never been incorporated into law. In the canton of Berne, Switzerland, however, we find the provision that every woman who has been employed on time rates in the same business for more than 1 year is entitled to 6 consecutive holidays on pay; after the second year's work, to 8 days; after the third, to 10 days; and after the fourth year, to 12 days.

Childbirth Protection.—The desirability of additional protection for working women at the time of childbirth has been recognized by most European countries and several outside of Europe. This protection consists of forbidding the work of women for a stated period, usually from 2 to 4 weeks before and from 4 to 6 or 8 weeks after childbirth. In Natal, South Africa, the prohibition applies for 3 months after delivery, and in Ticino, Switzerland, for 6 months after delivery. Only four American states—Connecticut, Massachusetts, New York and Vermont—have similar legislation. The prohibited period in Massachusetts and Vermont is 2 weeks before and 4 weeks after delivery, in Connecticut 4 weeks before and 4 weeks after, and in New York 4 weeks following delivery. It is doubtful if laws of this character are as necessary as in many European countries where the employment of mothers with young children is undoubtedly more common (see also "Insurance").

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Regulation of Eating Places.—Still another provision for the protection of women is that prohibiting the eating of meals in workrooms where processes are carried on which involve the use or presence of injurious or poisonous materials, dusts or fumes. Women and children in England are especially forbidden to take a meal or remain during meal hours in glass works where materials are mixed or where flint glass is made, polished, ground or cut; in earthenware works where dipping, drying or china scouring is done; where transfers are made; or where the tinning and enameling of metal hollow ware and cooking utensils are carried on. In addition, employees in such work must thoroughly wash their faces and hands before eating, and the employer is required to furnish adequate soap, towels, nail brushes and hot and cold water. Such restrictions are found in most countries and more often apply alike to both sexes. This is true in America, where the eating of meals in workrooms where certain poisonous substances, as lead, zinc or arsenic are used, is forbidden for all employees in New York, Illinois, Missouri, Pennsylvania, Ohio and New Jersey. In most countries or states where such prohibitions exist the employer is required to provide sanitary eating rooms for the convenience of employees.

Medical Examination.—Perhaps one of the most difficult things for an American to realize is the extent to which medical inspection of employees is practised in European countries. It is very commonly enforced for both men and women in the dangerous trades. We have seen that women and young persons are excluded from many of the more dangerous employments, particularly those embodying the use or presence of poisonous substances. In other occupations, where the workers are threatened with injury to health, women may not be employed unless after a thorough medical examination they are deemed to be physically fit. In many industries such examinations must be repeated at stated intervals.

In the decoration of china and earthenware, for instance, women in England must be medically examined at the beginning of employment and once each month thereafter. A health register is kept wherein is recorded the facts concerning the examination. If a woman is suspended from work she must be reexamined before she can return. In the enameling of iron plates, if any woman complains of illness which may be due to the nature of her work the employer must, at his own expense, give an order upon a doctor for immediate professional attendance. In this work it is recommended that light food, such as milk and biscuits, be supplied each woman before beginning work in the morning. Certificates of physical fitness are at times required before a woman may engage in night work or be permitted to work overtime.

In America while employers have at times voluntarily established medical examination of employees, yet special legal requirements for the medical examination of women do not exist. Women are, however, included with men in the laws requiring monthly physical examination of

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all employees engaged in dangerous processes in Illinois, Missouri, New Jersey, Ohio and Pennsylvania. These laws affect but few women, but should serve as a beginning for a much wider extension of this important preventive measure in the protection of women's health.

Insurance.—It has often been found that progressive employers provide more adequate protection for their employees than can be secured by direct legislation. An illustration of such a situation is found in the extensive preventive work which has followed upon the enactment of insurance or compensation laws. Such laws offer an economic inducement to prevent sickness or accident, and form, therefore, the third method of protection. These insurance measures apply with but few exceptions to men and women alike and are treated in detail elsewhere. In passing it may be said, however, that women are included in all of the health insurance laws of Europe —Germany, Austria, Hungary, Luxemburg, Norway, Great Britain, Russia, Roumania, and Serbia—and in the accident compensation laws of all civilized countries and of the 33 American states which by the end of 1915 had adopted accident compensation.

In addition to the ordinary sickness among industrial workers, women present a special problem—that is, illness at the time of childbirth. While in many cases, particularly among the working class of European countries, childbirth is a normal function causing disability for a short period only, yet all of these countries have recognized the desirability, from the point of view of the mother, the child and the state, of granting special financial aid at this time. In Germany and Norway the female wage worker is granted maternity benefits for 6 weeks, the period during which she is forbidden to return to work. Hungary follows the same time limit, while in Russia the period is only 4 weeks. Great Britain allows a lump sum of $7.20 to all insured women, regardless of marital condition, and $7.20 to wives of insured men, making a total of $14.40 for the insured wife of an insured man. Special mention must be made of the Italian system of maternity benefits established in 1910. This national compulsory system applies to all women wage earners in factories between the ages of 15 and 50, and applies alike to married and unmarried workers. It is supported by contributions from the state and from the employer and employee who contribute in equal amounts. The total amount of the benefit given is $7.72, which in Italy aids greatly in assuring proper care of both mother and child at this critical time. Although several American states have already prohibited the industrial employment of women for a period before and after childbirth, yet no form of health or maternity insurance exists by law in America, although many voluntary societies provide such insurance for their members.

It is apparent that in the United States considerable progress has been made toward adequate protection of the health of working women, yet in many respects American legislation lags far behind the legal protection

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already afforded by leading European countries to the women who, with slighter physique, are competing with men amid the special health hazards of modern industry.

REFERENCES

[1] For an excellent statement of the world's experience in the effect of industry upon the health of working women see Fatigue and Efficiency, by Josephine Goldmark, Russell Sage Foundation, New York City.

[2] Lead Poisoning in Potteries, Tile Works, and Porcelain Enameled Ware Factories, Alice Hamilton, M. D., U. S. Bureau of Labor Statistics, Bulletin No. 104.

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CHAPTER IV

THE EXCLUSION OF MINORS FROM INJURIOUS AND DANGEROUS OCCUPATIONS

BY OWEN R. LOVEJOY, New York City, N. Y.

Professor Teleky of Vienna University has lately published his findings as to the effect of work on the health of wage-earning children. His study showed especially the susceptibility of working children to tuberculosis, but he found, too, that the general sickness rate in a given group of children increases after they have left school for work. The increase continues so that in a period of 4 years it is greater the fourth year than it was the first. That is, the wear and tear of industrial life tells on the children and produces in them physical ills that grow greater rather than less as times goes on.

We have all felt that something like this was true, but we have not often succeeded in getting it down in black and white as Professor Teleky has done, and even with actual figures before us it is difficult to realize their full import. To appreciate the physical effect of industry on working children we must see the children; we must regard our data not merely as statistical but as real moulding forces in their lives; we must realize what it means to John Smith, a normal small boy of 14, to work as a doffer in a cotton mill for a year and there contract severe bronchitis with a cough, anæmia, and cotton fibroid pthisis so that at 15 he is far below normal and handicapped. Nothing can so vivify for us the case of the working child as do the mere unvarnished facts published by public health authorities. We find, for instance, reports of such occupational injuries as these:

"Fred ———, age 14, employed by ———, manufacturers of picture frames. Worked putting bronze on frames. Produced severe conjunctivitis of the eyes; throat coated with bronze dust; expectoration of green bronze matter.

"John ———, age 15, employed by ———, enameling and stamping company. Boy handled grease and enamel. Occupational dermatitis, severe irritation of skin of hands and arms.

"James ———, age 15, employed by ———, chair factory. Worked sandpapering woodwork adjacent to jointers and stainers where lead enamel was used, the fumes of which produced symptoms of headache, gastric pains and nausea, sore and pale gums; teeth affected; became pale and weak; metallic lead breath.

"Joseph ———, age 15, employed by ———, opticians, as apprentice boy. Worked drilling and cementing in which wood alcohol is used. Fumes made boy sick, producing nausea and vomiting, asthma, insomnia, pupils unevenly dilated.

"Edward ———, age 14, employed by ———, cigar makers, as errand boy. Fumes of tobacco produced symptoms of headache, sick stomach. He became weak, with dilated pupils and tobacco pallor—a clear case of nicotine poisoning."

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In another report we read of other injuries to children, this time due to machine accidents.

"F. S., male, 14, nail sticker, earning $4 a week operating American lightening heeling machine. A nail flew out of the loader as nails were released. Injured boy attempted to brush it off with his finger. He was caught by descending drivers. Bone broken in forefinger of left hand between first and second joints. Flesh torn and cut.

"F. K., male, 15, earning $3.50 a week operating baling press. Gearing wheels carefully guarded. Notwithstanding, he stooped and placed his hands under the guard to the gearing wheels. Lacerated first finger, amputation second and third finger at the first joint, lacerated middle finger.

"M. C., female, 14, earning $3 a week, operating automatic cutting machine. Was waiting for work to be given her and took screw driver and was scraping around with it; she put her foot on the starting lever and drew in the screw driver, also her finger. First finger of left hand was smashed and apparnetly broken and was later taken off at the first joint."

These are but a few of the many cases reported, but they force us to recognize the hazards of industry for the working child, and we must regard those hazards as all the more serious when we realize that after all in the cases here cited the children were not working at what are generally classified as dangerous trades. They were working at occupations which we, in our present state of enlightenment, have considered safe. To be sure, we have recognized lead poisoning as one of the most insidious of industrial diseases, but even where we have forbidden children to work at trades in which lead poisoning is probable, we have not realized that a child working near jointers and stainers using lead enamel may in a comparatively short time be affected by the poison. In the same way, although in our most advanced legislation we have forbidden children to operate recognizedly dangerous machines, we have not realized that machines apparently safe may become dangerous in the hands of the untrained and immature.

Until very recently our efforts to protect the health of working children here in the United States have been strangely scattered and unscientific. Now with a Federal Children's Bureau, an increasing number of public health boards and growing interest in industrial hygiene we seem to be embarked on a new era. The value of the public health board cannot be overestimated. The mere recital of the subjects the State Board of Health in Massachusetts, for instance, was required to investigate suggested the scope and power of the board. Among the subjects were:

1. The prevalence of diseases dangerous to health in home, schoolhouse, factory or elsewhere.

2. Sanitation of schoolhouses and industrial establishments.

3. Information concerning the health of young persons in factories at their work, and the influence of such occupation upon the health of these minors.

With such a program it is obvious the board of health could make tremendous advances in the direction of the better understanding and protection of

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the child in industry and of the conservation of public health in general. But, unfortunately, the Massachusetts Board of Health was handicapped, as Dr. William C. Hanson has pointed out, by the lack of proper places for the examination of young persons and by "the absence of authority to exclude from factories young persons found in ill health or physically unfit."*

This situation is typical of our carelessness and laxness in legislating for the protection of public health. We provide for public health officers, but we do not give them power enough. We aim to protect the child from industrial hazards, but we do not fully protect the child. The essential point of both Professor Teleky's conclusions and the quoted medical reports, that the organic differences between the child and the adult make safeguarding the working child an entirely different problem from safeguarding the working adult, is something we have not sufficiently emphasized. If the child is peculiarly susceptible to industrial strain and so to industrial injury, we must make drastic, special regulations to prohibit the child from working under conditions that will inevitably lead to physical injury. This already recognized principle has not yet been carried far enough to be effective. We have considered it in connection with age limits and hours of work, but in what seems to be its most obvious application we have not made it effective. In legislating for the exclusion of children from dangerous trades we have been strikingly lax.

There are at present 26 states in the Union that make some provision for the exclusion of children under 16 from dangerous trades, and three other states besides 12 of the 26 specify other dangerous trades in which persons under 18 may not be employed. Fourteen states prohibit the employment of persons under 21, or minors in a few other trades injurious to morals as well as to health, or affecting the safety of others. But in most cases the statutes are vague, there is no uniformity in them. Where one state specifies simply mines and quarries as dangerous, the next gives a long list of carefully defined processes or occupations, and a third prohibits the employment of children "in any occupations dangerous to health or morals," leaving the definition of such occupations to the enforcing officials, and it is worth while here to note that almost invariably when a state has such a loose statute, the machinery for its administration is so weak that the law is practically a dead letter.

The only attempt at uniformity in such legislation in the United States is the "Uniform Child Labor Law" drafted for the National Conference of Commissioners on Uniform State Laws in 1911 by the National Child Labor Committee and later endorsed by the American Bar Association. This law, which embodies the best provisions in various state laws, has seven sections† dealing directly with dangerous trades. Children under 16 are prohibited from working in the more obviously dangerous trades, such as in operating dangerous machinery, on railroads and vessels, in occupations in, about or

* The Massachusetts State Health Officers no longer have legal authority to examine the health of young persons in factories. [Ed.]

† The text of these seven sections is given at the end of this article.

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in connection with poisonous acids, dusts or gases, in heavy building trades, in mines or quarries, in bowling alleys, pool or billiard rooms, or on the stage. The State Board of Health is empowered to determine from time to time whether any processes or occupations not specified are dangerous to the health of children and to make necessary prohibitions. Children under 18 are forbidden to work in or about blast furnaces, docks or wharves, in certain electrical work, in cleaning machinery in motion, in connection with specified machines, in definitely specified railroad and navigation work, in connection with processes in which explosives or phosphorus are used, and in any distillery, brewery or place where alcoholic liquors are sold. The State Board of Health is again empowered to determine whether other occupations not specified are dangerous to these children and to forbid their employment where necessary. No person under 21 may be employed in a saloon or barroom where intoxicating liquors are sold, and no female under 21 may work in mines, quarries, coal breakers, or in oiling or cleaning machinery in motion, or where she is compelled to stand constantly.

This is an inclusive, strong law, not drastic enough to make its passage difficult in most states. Several states have laws practically identical with sections of it and since it is based on state laws it contains nothing entirely new. It is decidedly the best law we have to offer here in America, but even in advocating it, we should not regard it as ideal but simply as the first step toward something better. Probably its best feature is that it leaves room for constant improvement by providing that the State Board of Health may from time to time determine whether any processes not specified in the law are injurious to the health of children. With our regulation of dangerous trades in its present embryonic state there must be plenty of room for such improvement, but even if we were sure we had docketed and pigeonholed all dangerous elements in industry the more elastic the law the better. The best law will rather name the dangerous element in the work than the specific process or trade, so as to permit of application to any process in which the dangerous element occurs, and the Public Health Board must be given power to make the application.

But there are bound to be questions as to the definition of dangerous trades which must sooner or later be answered. In some states, for instance, work in mines and quarries is not recognized by law as dangerous. Yet in Minnesota between 1909 and 1912 there were 224 accidents in mines as compared with 112 in lumbering and woodwork, 69 in contracting, and smaller numbers in other industries. If these figures are representative, obviously mining is a hazardous trade, but not until accident reporting is made effective can we expect to know what are really hazardous trades. For the present it is safe to say that if a trade is so dangerous that we have to take special precautionary measures to safeguard adults engaged in it or have provided special compensation or medical inspection for the workmen, or if accidents

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are known to be frequent, then that trade is one from which children must be excluded.

But the question is, who are "children?" It is necessary to fix an age limit under which young persons may not be employed in hazardous occupations. The Uniform Child Labor Law represents our recognized standard of age limits. It is based on the principle that no children under 14 may enter industry and that no children under 16 may enter any hazardous industry. Extra-hazardous trades it prohibits to children under 18 as well as any trades in which the safety of others is dependent on the worker, and which have a morally injurious effect. It prohibits certain other trades which fall under these last two classes to persons under 21, and recognizes the physical differences between men and woman by making special prohibitions for women under 21.

Reasons for these age limits are easily understood: They represent our ingrained ideas of the comparative strength and ability of children between the ages of 14 and 21, but there is no reason for believing that those ideas are final. In fact there is a growing feeling that children should not be in any industry at all until they are 16. Professor Teleky drew that conclusion from the facts he discovered. He believes that 16 is young enough for any child to be employed, and he advocates the strict limitation of hours of work for children under 18 and the provision for education and recreation for such children. At a hearing of the United States Commission on Industrial Relations in New York in May, 1914, several witnesses testified to their belief that children should not be employed before they are 16. The head of the hygiene department of the New York City Board of Health, who carries on the physical examination of children seeking employment certificates, stated emphatically that children of 14 were not physically fit for industry. The chief mercantile inspector for New York City said that his experience and the testimony of merchants with whom he came into contact made him certain that children of 14 were not mentally capable of doing the work required of them in mercantile establishments, and that the physical burdens they had to bear were usually too much for them. If it is becoming apparent, as it seems, that children should not work at ordinary trades until they are 16, then surely the age limit for employment in hazardous trades must be 18 at least, and there is a great deal to be said for the prohibition of all dangerous trades to persons under 21.

Professor Teleky's report shows that the evidences of industrial strain on children increase rather than decrease as time goes on. Some of this increase is, of course, due to the cumulative effect of strain, but some of it, too, must be due to the fact that the children are still in the process of growth which makes them susceptible to injury. Most children probably do not "get their growth" and wholly adapt themselves to full growth before they are 21. Therefore persons under 21 may be considered as organisms still in the process of growth and so unfitted for extraordinary industrial hazards,

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and it seems unnecessary and contrary to the final economy of public health to expose the immature to the dangers of hazardous occupations.

It is this final economy that is our modern concern. The exclusion of the immature from dangerous trades is the logical consequence of the present conviction that we must scientifically conserve human resources. It is not merely "humanitarian;" it is a scientific necessity. The need for the exclusion of the immature from injurious work, for the medical supervision of all workmen and for careful physical examinations for all children both before they go to work and while they are at it cannot be overemphasized in this country. Until such medical supervision is made general we shall not have really gripped the problem of industrial hygiene in the United States.

In the meantime, it is imperative that the provisions of the Uniform Child Labor Law, or their equivalent, be enacted in every state in the Union, and that occupational hazards for children be studied and defined by public health agencies. The public must put an end to incompetence or self-interest in the public health service. Medical associations, state boards of health, physicians and experts in hygiene—those who best know the facts—must serve as pioneers in demanding that the public health department shall be elevated to its rightful place and be made the most important arm of city, county or state government. It should be impossible in the United States for a child of 15 to contract lead poisoning or wood alcohol blindness. Even John Smith with his bronchitis, anæmia and cotton fibroid pthisis should be an exceptional case and not one of many, as he is to day.

Text of sections of the Uniform Child Labor Law:

Section 3.—No child under the age of 16 years shall be employed, permitted or suffered to work at any of the following occupations or in any of the following positions: Adjusting any belt to any machinery; sewing or lacing machine belts in any workshop or factory; oiling, wiping or cleaning machinery or assisting therein; operating or assisting in operating any of the following machines: circular or band saws, wood shapers, wood jointers, planers; sandpaper or wood-polishing machinery; wood-turning or boring machinery; picker machines or machines used in picking wool, cotton, hair or any other material; carding machines; paper-lace machines; leather-burnishing machines; job or cylinder printing presses operated by power other than foot power; boring or drill presses; stamping machines used in sheet metal and tinware or in paper and leather manufacturing, or in washer and nut factories; metal or paper cutting machines; corner staying machines in paper-box factories; corrugating rolls, such as are used in corrugated paper, roofing or washboard factories; steam boilers; dough brakes or cracker machinery of any description; wire or iron straightening or drawing machinery; rolling mill machinery; power punches or shears; washing, grinding or mixing machinery; calendar rolls in paper and rubber manufacturing; laundering machinery; or in proximity to any hazardous or unguarded

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belts, machinery or gearing; or upon any railroad, whether steam, electric or hydraulic; or upon any vessel or boat engaged in navigation or commerce.

Section 4.—No child under the age of 16 years shall be employed, permitted or suffered to work in any capacity in, about or in connection with any processes in which dangerous or poisonous acids are used; nor in the manufacture or packing of paints, colors, white or red lead; nor in soldering; not in occupations causing dust in injurious quantities; nor in the manufacture or use of dangerous or poisonous dyes; nor in the manufacture or preparation of compositions with dangerous or poisonous gases; nor in the manufacture or use of compositions of lye in which the quantity thereof is injurious to health; nor on scaffolding; nor on a ladder; nor in heavy work in the building trades; nor in any tunnel or excavation; nor in, about or in connection with any mine, coal breaker, coke oven, or quarry; nor in assorting, manufacturing or packing tobacco; nor in operating any automobile, motor car or truck; nor in a bowling alley; nor in a pool or billiard room; nor in any other occupation dangerous to the life and limb, or injurious to the health or morals of such child; nor shall any child under the age of 16 years be employed upon the stage of any theatre or concert hall or in connection with any theatrical performance or other exhibition or show.

Section 5.—The state board of health may, from time to time, determine whether or not any particular trade, process of manufacture or occupation, in which the employment of children under the age of 16 years is not already forbidden by law, or any particular method of carrying on such trade, process of manufacture or occupation, is sufficiently dangerous to the lives or limbs or injurious to the health or morals of children under 16 years of age to justify their exclusion therefrom. No child under 16 years of age shall be employed, permitted or suffered to work in any occupation thus determined to be dangerous or injurious to such children.

Section 18.—No child under the age of 18 years shall be employed, permitted or suffered to work in, about or in connection with blast furnaces, docks or wharves; or in the outside erection and repair of electric wires; in the running or management of elevators, lifts or hoisting machines; in oiling or cleaning machinery in motion; in the operation of emery wheels or any abrasive, polishing or buffing wheel where articles of the baser metals or iridium are manufactured; at switch tending, gate tending, track repairing, or as brakemen, firemen, engineers, motormen or conductors upon railroads, or as railroad telegraph operators; as pilots, firemen or engineers upon boats and vessels; or in or about establishments wherein nitroglycerine, dynamite, dualin, guncotton, gunpowder or other high or dangerous explosives are manufactured, compounded or stored; (13) or in the manufacture of white or yellow phosphorus or phosphorus matches; (14) or in any distillery, brewery, or any other establishment where malt or alcoholic liquors are manufactured, packed, wrapped or bottled; (15) or in any hotel, theatre,

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concert hall, place of amusement, or any other establishment where intoxicating liquors are sold.

Section 19.—The state board of health may, from time to time, determine whether or not any particular trade, process of manufacture or occupation, in which the employment of children under 18 years of age is not already forbidden by law, or any particular method of carrying on such trade, process of manufacture or occupation, is sufficiently dangerous to the lives or limbs or injurious to the health or morals of children under 18 years of age to justify their exclusion therefrom.

No child under 18 years of age shall be employed, permitted or suffered to work in any occupation thus determined to be dangerous or injurious to such children.

Section 20.—No minor under 21 years of age shall be employed, permitted or suffered to work in, about or in connection with any saloon or barroom, where intoxicating liquors are sold.

Section 21.—No female under 21 years of age shall be employed, permitted or suffered to work in or about any (1) mine, (2) quarry, (3) or coal breaker, except in the office thereof, (4) or in oiling or cleaning machinery while in motion.

Section 22.—No female under 21 years of age shall be employed, permitted or suffered to work in any capacity where such employment compels her to remain standing constantly.

Every person who shall employ any female under 21 years of age in any place or establishment mentioned in Section 1 shall provide suitable seats, chairs or benches for the use of the females so employed, which shall be so placed as to be accessible to said employees; and shall permit the use of such seats, chairs or benches by them in so far as the nature of their work allows, and there shall be provided at least one seat to every three females.

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CHAPTER V

THE WORK OF LABOR AND HEALTH BOARDS AND OF LEGISLATIVE COMMISSIONS

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The General Problem.—To describe and estimate the value of measuring public administration in relation to the combat with occupational diseases. The coöperative effort to reduce these evils requires: (1) the avancement of science in this field; (2) the popularization of knowledge; (3) the expression of the enlightened judgment of the public in the form of law; (4) the establishment and maintenance of suitable, competent and efficient administrative organization.

The Scope of This Chapter.—Administrative organization and its activities is the special problem of this chapter, which includes (1) federal, (2) state, and (3) local agencies.

1. FEDERAL ACTIVITIES

The activity of federal authorities in the interest of public health rests chiefly on the clause in the constitution which gives Congress considerable power over the operations of interstate and international commerce, but the clause relating to the common welfare has also had great influence. The investigation of diseases, the production and distribution of vaccines, the laboratories for the analysis of food stuffs and medicines, and the publication of bulletins of information go beyond mere commercial regulations.

By an act approved August 14, 1912, the name of the "Public Health and Marine Hospital Service"[1] was changed to the "Public Health Service," and this branch was authorized "to study and investigate the diseases of man and spread thereof," and to publish information for the public. By an act approved August 23, 1912, Congress authorized the appointment of a temporary "Commission on Industrial Relations," one of whose duties is to inquire into matters relating to the health of employees.

By an act of Congress (approved April 9, 1912) the use of poisonous white phosphorus matches was abolished from our country by laying a prohibitive tax of 2 cts. per 100 matches.

The establishment of the Childrens' Bureau (act approved April 9, 1912) was a significant and prophetic action.

* Deceased.

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Another law enacted in 1912 evidently rests partly in regard for the health of workmen, since it forbids contractors on federal contracts to keep men at work longer than 8 hours in 1 day.

The Public Health Service, with its central bureau at Washington, has as its head the Surgeon-General, with an Assistant Surgeon-General over each of its seven divisions: personnel and accounts, foreign and insular quarantine and immigration, interstate quarantine and sanitation, sanitary reports and statistics, scientific research, marine hospitals and relief, miscellaneous. Up to this time this service has been chiefly occupied with fighting communicable diseases and epidemics and caring for seamen. The bureau has a corps of 450 medical officers, 50 pharmacists, with a total personnel of about 2000 persons. It seems probable that in the future the scientific investigations of this service may be extended into the field of industrial hygiene; in scientific investigation it has already advanced knowledge of the subject.

The United States Bureau of Labor is an indispensable factor in the combat with industrial diseases.[2]

Mr. Verrill, of this Bureau, has made an official statement of the function of his office which is cited here:

"Unlike the state factory inspection departments and the state boards of health, the Federal Bureau of Labor is not charged with the duty of enforcing or formulating regulations. Its functions are limited to the work of investigation and study and the diffusion of information, but with these limitations—that the duty is imposed on the Bureau of studying the means of promoting the material, social, intellectual, and moral prosperity of the working men and women. It is quite within its scope, therefore, to study the subjects of accidents with a view to the elimination, so far as possible, of dangerous machinery and working conditions, and the introduction of protective devices, or to the study of factory processes and conditions with a view to eliminating, restricting, or safeguarding the use of poisonous and injurious materials, or to study lighting, ventilation, humidity, the disposal of dust or fumes, and the improvement of the hygienic conditions of the working places.

"In the group of reports relating to factory inspection and occupational hygiene, are included articles in regard to the inspection of factories and workshops in this and in foreign countries. The details in regard to factory legislation and inspection, the regulation of working conditions, especially in industries and occupations involving special dangers, have been the subject of special attention. Special studies have also been made of the mortality and morbidity in certain dangerous occupations and of the dangers to employees who, in the course of their work, come into contact with certain poisonous materials. Under this head, of the greatest importance are the studies of phosphorus poisoning in the match industry and of lead poisoning in the lead industry and in the manufacture of pottery, tiles, and porcelain

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enameled sanitary ware. In all of these industries investigation has disclosed the existence of dangerous conditions, most of which may be entirely eliminated, or very greatly improved, without serious difficulty. All of these investigations have strongly emphasized the fact that, by a study of working conditions in the more dangerous and unhealthful occupations, the methods and experience of the best may be available for those factories where, because of ignorance or because of indifference resulting from ignorance, dangerous conditions have been allowed to continue without any technical necessity and without any economical need." The Supreme Court of the Union[3] has become a very vital factor in securing, interpreting and supporting desirable legal requirements for the protection of the health of workmen. There is a notable and encouraging tendency of the best courts to confirm legislation which is not merely based on "matters of common knowledge" but on scientific authority which is always in advance of "common knowledge" and much more exact and reliable. It is evident that henceforth the studies and judgments of experts, where there is a reasonable measure of agreement, are to be made the foundation of legislative and of judicial interpretations of state and federal constitutions. In this connection the "legislative reference bureau" of Wisconsin is a sign of promise, because the information there gathered will help to frame laws which rest on that foundation which will later endure the critical examination of the highest judicial authorities, and exactly the same facts which guided lawmakers will be available when the test cases are tried. The medical men and engineers have here a new and promising field of service to mankind.

Dr. Devoto, of the Italian hospital for occupational diseases at Milan, generously and wisely showed the intimate and reciprocal relations of medical and social science, when he said: "In 1898 I had the good fortune to read that which the learned and eminent sociologist, Hector Denis said, with his clear intelligence: 'Physician! Come forth from your limited domain; go to the workingmen, and in labor you will find an immense field of study.'"[4]

2. STATES

Legislation: States.—The fundamental legal requirements relating to occupational diseases are found in the statutes of the state and of the federal government, as interpreted, in case of disputes by the courts. These statutes rest upon "police power" of government; that is the exercise of public authority "to secure the public welfare" by restraint and compulsion (E. Freund, Police Power, pages 3, 7). The laws passed by legislatures in the U. S. are collected in the 22d Annual Report of the Commissioner of Labor (1907), Labor Laws of the U. S., supplemented by later bulletins.

It is not within the scope of this chapter to analyze these laws relating to ventilation, light, dust removal, etc., except so far as concern the powers and duties of officials charged with enforcement of those laws.

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Administration.—1. State Boards: aim, scope, powers, duties.

There are two types of organs which deal with the health, safety and comfort of workers: labor boards and health boards.

(*a*) *Labor Boards.*—This is the prevalent type. It is found in many states of the union. "In many of the states having laws of this class provision is made for their enforcement by means of special officials or inspectors, as labor bureaus, factory inspection officers, and mine bureaus; while in others this duty devolves upon such officers as are charged with the enforcement of the laws generally.

"It need hardly be added that in states of the latter class the laws are usually inefficiently enforced. The laws of the various states differ in their nature, some being absolute or mandatory in form, directing certain provisions to be made under prescribed conditions, while others commit large discretion to the inspecting as enforcing officers. The latter laws are open to criticism as affording opportunity for a variety of standards, as the judgment and disposition of the enforcing officials may vary. A law that provided that, if it appeared to the enforcing officer that the . . . conditions could, to a great extent, be prevented by the use of some mechanical contrivance, he should direct that such contrivance be installed (Cal. Act of Feb. 6, 1889) was declared void on the ground that it imposed on the inspector, not the duty of enforcing the law of the legislature, but the power of making a law for the individuals, and enforcing such rules of conduct as he might prescribe, which was an unconstitutional delegation of legislative power (Schaezlein vs. Cabaniss, 135 Cal. 466, 67, Pac. 755). At what point the line would generally be drawn by the courts is not clear, since much of the detail must of necessity be left to the judgment and integrity of the enforcing officers; and such expressions are quite common as 'in the discretion of the chief inspector' (Ind. A. S., Sec. 7087, I), 'as the factory inspector may direct' (Conn. Acts, 1905, Sec. 13), 'if it appears to the inspector that such (injurious inhalation would be substantially diminished' (Mass. Acts, 1909, Ct. 514, Sec. 84); impossible" (Arms and Ayer, 192, III, 601, 61, N. E. 851; St. Louis Crusol Coal Co., v, III, 185, U. S., 293, 22, Sup. Ct. 616). The Wisconsin system is instructive at this point.

Illustrations may be given of a few of the more elaborate laws touching industrial dangers.

Illinois.—One law requires the use of blowers on metal polishing and grinding machinery. The "Health, Safety and Comfort Act" provides for adequate ventilation, both artificial and natural, lavatories for both sexes, the disposal of noxious fumes, gases and vapors, proper temperature of working rooms, seats for women. The "Occupational Disease Act" (in force July 1, 1911) requires employers to adopt reasonable and approved devices for the prevention of occupational diseases, especially when workmen are exposed to poison by lead or other poisonous substances, or in the manufacture of brass or the smelting of lead or zinc. These provisions were adopted

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as a result of the studies and report of the Commission on Occupational Diseases.

The chief factory inspector and his staff are charged with the enforcement of the laws and with the prosecution of all violations before any magistrate or any court of competent jurisdiction in the state. The penalty for violating the law or obstructing the inspectors is a fine for the first offense of $10 to $50; for later offenses $25 to $200. When the inspectors discover a violation of the law they are to notify the managers. The essential requirements of the law must be posted up in various languages in all establishments covered by the provisions of the act.

The department of inspection employs a physician and a physician-dentist, both of whom are employed daily in the making of personal inspections under the "Health, Safety and Comfort Act" and the "Occupational Diseases Act." The medical men are engaged in original research; they have studied, for example, the effect of lead sulphate on animals (Letter of Oscar F. Nelson, chief inspector, Sept. 8, 1913). The law requires any licensed physician who makes the physical examination (which is required when men are brought into contact with poisonous agencies or injurious processes) to make an immediate report to the state board of health, and this board sends a copy to the department of factory inspection. If the employers neglect the required protective arrangements, an injured party or his family has a right of action for damages not to exceed $10,000.

Michigan.[5]—The governor is authorized to appoint a commissioner of labor, for 2 years. This commissioner of labor may appoint factory inspectors and assistants; his duty is to report on the "moral and sanitary conditions of the laboring classes and the productive industries of the state," and he is to inspect all work places.

The law restricts hours of women and children; forbids unsuitable employments; requires exhaust fans to remove dust; toilet rooms and wash rooms are to be provided; tenement house work is regulated; seats for women are required; ventilation of foundries is specified; sanitary regulations for mines are given.

Complaints of neglect of law may go to a justice of peace or magistrate. Local boards of health are to report to inspectors cases of infectious or contagious diseases.

Missouri.—An "Occupational Disease Law" has been in force since June 23, 1913. It is the duty of licensed physicians to report cases of such diseases to the state board of health in duplicate; one copy is sent to the factory inspector and one to the superintendent of the factory. Various hygienic regulations (wash rooms, clothing, separate eating places, drinking water) are included in the law. It is the duty of the state factory inspector to inspect and enforce the regulations, and prosecute violations. The employer must post warning notices of danger.

Maryland.—A state department of health (1912) exists. The law (Ch.

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165, Laws of 1912) describes occupational diseases; requires physicians to report to the state board of health any cases known to them. It is the duty of the state board of health to enforce the law; it may call on local boards of health and health officers for aid; and it must report date to chief of Maryland bureau of statistics and information to be published in annual report.

New York.[6]—The department of labor is under the commissioner of labor, who is appointed by the governor by and with the advice and consent of the senate, with a term of 4 years in office. The commissioner appoints all officers, clerks and other employees in the office. There are three bureaus in the department of labor: factory inspection, labor statistics, mediation and arbitration. The bureau of factory inspection includes the chief factory inspector (first deputy commissioner), 1 assistant, mechanical engineer, superintendent of licenses, medical inspector of factories, 2 tunnel inspectors, mine inspector, 8 supervising factory inspectors, 85 factory inspectors, 1 chief clerk and 20 stenographers and clerks. One medical inspector of factories in a great industrial state like New York is manifestly at a disadvantage. His report for 1912 shows an effort to study various industries with a view toward classification of those of a dangerous nature, and the investigation of cases of occupational poisoning reported, with the hope of discovering the causes and applying a remedy. Chemical industries and color works have been investigated. Laboratories and clinics have cooperated in the study of lead and arsenical poisoning. Hygienic exhibits have been prepared for public instruction. A mechanical engineer was employed to recommend better methods of ventilation of shops and the removal of dust, fumes, gases and vapors.

Ohio.—The state board of health, beginning May, 1913, has been conducting a "survey of occupational diseases" to secure a basis for future legislation and administration. This survey has been conducted by E. R. Hayhurst, M. D., who was one of the medical investigators of the Illinois Commission on Occupational Diseases, under the authority of the board. The survey is conducted by means of articles published in the monthly bulletin and in newspapers, by lectures and traveling museum exhibit; by distribution of blank forms among physicians for reports of cases of occupational diseases; by medical inspection of industrial establishments to discover cases; and by experimental work under the auspices of the Ohio State University. Physicians are required by law to report cases of poisoning from lead, phosphorus, arsenic, brass, wood alcohol, mercury or their compounds, contracted as a result of the patient's employment, within 48 hours from the time of first attending such patient (law passed March 25, 1913). The board of health has considerable discretion in relation to the forms of reports and the kind of information demanded; "such information as may be reasonably required by the state board of health." This is a special investigation conducted by a regular and permanent state organization.

The Ohio state board of health has large legal powers: supervi-

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sions of all matters relating to the preservation of the life and health of the people. "It may make special or standing orders or regulations for preventing the spread of contagious or infectious diseases . . . . and for such other sanitary matters as it deems best to control by a general rule." Violations of laws or regulations are prosecuted in the courts by customary procedure.

Provisions are made for local boards of health in cities and villages. The trustees of each township constitute the board of health and they must appoint a health officer.

Pennsylvania.—An act (taking effect June 1, 1913) contains the following provisions. The department of labor has for its head the commissioner of labor and industry, appointed by the governor, with consent of the senate. The bureau of inspection has inspectors of four grades, and of these some must be physicians, with a chief medical inspector. A medical engineer, a chemical engineer and a civil engineer belong to the corps of inspectors. The inspectors of the fourth grade shall constitute a division of industrial hygiene, which shall be under the immediate charge of the commissioner.

Massachusetts.—Until recently Massachusetts placed the health interests of the working people under the supervision and authority of representatives of the medical profession. The state board of health formerly dealt with hygienic control of the shops as a part of the hygiene of the community. The same agency which investigated the dangers of occupations was seeking to control the evils which arise in the home and community life of the worker. The state inspectors of health were in constant communication with local physicians who could give information concerning not merely communicable but also occupational diseases (Paper of W. C. Hanson, M. D., 15th International Congress of Hygiene and Demography, Vol. III, page 898).

The principles upon which the state board of health was proceeding have thus been stated by one of their inspectors (15th International Congress on Hygiene and Demography, Vol. III, Dr. H. Linenthal, State Inspector of Health, Boston, page 907). To summarize briefly: For the prevention of the ill effects upon health from industrial processes or conditions, the following measures should be adopted:

1. To collect complete and accurate data about industrial processes and about conditions under which the various industries are carried on.

2. To obtain more accurate and detailed information relative to occupation on morbidity and mortality records.

3. To instruct the medical student in this important field of preventive medicine by a course of lectures on the more important industrial processes and the diseases to which they give rise.

4. To place the specific industrial diseases on the list of diseases notifiable to the central health authority.

5. To examine periodically all workers in certain industries. These industries should be named by the central health authority.

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6. To exclude minors and women from certain industries which are designated by the central health authority as injurious to health.

7. To have adequate laws regulating sanitary conditions and protective devices in industrial establishments, and to have such laws intelligently enforced.

8. To have the central health authority issue regulations for certain dangerous trades, with instructions to employers and employees, showing them how they may guard themselves against the ill effects of their work, and to have such instructions posted in the workrooms.

9. To carry on an extensive educational campaign, both among employers and employees as to the value of protective measures and good sanitary conditions.

By the law of June 10, 1912, the inspection of health conditions in shops was placed under an entirely different control. ("An act to establish a state board of labor and industries.") The new board is composed of one employer, one laborer, one physician and one woman. This board appoints a commissioner of labor and retains power to remove at any time. It has power to investigate conditions, hold hearings, seek expert advice and prosecute violators of the law. The duties of the boards of health and of police, so far as they related to industries, have been transferred to this new body which may appoint industrial health inspectors and assistants. The new industrial health inspectors are not legally required to have a medical training. The medical inspectors under the state department of health no longer enforce the law relating to health inspection in factories. They have, however, opportunity to enter factories, if the state commissioner of health deems it necessary.

(*b*) *The Industrial Commission of Wisconsin.*[7]—The Wisconsin law differs from other laws by lodging in a state commission considerable legislative authority, but guards against abuses in various ingenious ways. The state law expresses the general will as to the duty of the employers to protect the life, health and safety of the employees and the duty of the latter to coöperate in carrying out the purpose of the law. The Industrial Commission is charged with making this general law effective. It calls in scientific experts in hygiene and engineering to establish standards for regulation of all places where productive processes are carried on. These regulations must be "reasonable." In making the orders, which have the form of law, employers and employees have a right to be heard, according to their special knowledge and interests. The procedure is somewhat slow, but when regulations are drawn they express the convictions of the persons affected, who are also most intelligent in regard to the practicability of the requirements proposed by the scientific advisers.

(*c*) *Temporary Commissions of states,* created by the legislature for a particular exploration, with reference to legislation on the subject.

In states where the great industry has not been developed and the evils of accident and disease have not been vividly felt, and where boards of health and of inspection have not been established; or where they are incompetent or neglectful, a special temporary commission may render a valuable service.

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An investigation carried on by legislative authority carries more weight than one instituted by private students, and their researches issue in practical bills for laws which give permanence to the results.

The General Assembly of Illinois in May, 1905, authorized the governor to appoint a commission[8] to investigate the subject of social insurance and workingmen's old age pensions and to report the draft of a bill. This commission reported in 1907, but the time was not then quite ripe for insurance legislation; later the study bore fruit in a compensation law. In this report that commission urged the legislature to create another commission to study occupational diseases. The governor was authorized to appoint a commission of nine members, including the state factory inspector, the secretary of the bureau of labor statistics, the president and secretary of the state board of health, two reputable physicians and three other citizens. This commission organized a corps of investigators, made studies especially of poisoning by lead, arsenic, mercury, gas in foundries, caisson diseases, and miners' exposure to disease. The report was laid before the legislature in January, 1911. As an immediate result the legislature extended the protective measures to include shop hygiene (House Bill, No. 250, approved May 26, 1911). In Illinois court decisions and tradition have rigidly restricted the power of the legislature to delegate its functions to administrative bodies; and therefore it was necessary to make the law explicit in all the regulations which the factory inspectors were required to enforce. In this respect the Illinois method is radically different from that of Wisconsin; and yet the inspectors were required to act in interpreting what were "reasonable and approved devices" for the prevention of occupational diseases. Periodical medical examination of workmen exposed to lead poisoning was required, and this clause introduced a new principle into the law. Medical men were for the first time added to the staff of inspectors; a laboratory was established; and this factor has been found helpful in detecting and averting danger to health.

The New York Factory Investigating Commission was another example of a temporary organization for the exploration of conditions. Its appointment was due to a tragic loss of life in connection with the burning of a work place. Engineering problems were primarily considered.

Local Health Organization—Municipal.[9]—The primary function of municipal health boards and commissioners, according to ordinances published, are interpreted to be those relating to the suppression of nuisances; the combat with communicable diseases; notification, isolation, disinfection; examination of school children; vaccination; control of barber shops, street cars, lodging houses and tenements, laundries, wash houses, common drinking cups, rags and secondhand goods, food stuffs, milk and milk products, housing and plumbing, privies and cesspools, stables and disposal of manure, domestic animals and dead bodies, garbage, refuse and ashes, offensive trades, midwifery and lying-in houses, births, marriages and interments—that

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is, with matters of general concern. Only in exceptional regulations do we discover as yet any distinctly conscious purpose of municipal authorities to study and supervise industrial establishments and the conditions affecting the health of workmen.

An interesting illustration of local voluntary regulation of conditions affecting health is the organization in the garment-working trades of New York City, where 60,000 to 70,000 persons are engaged in a business whose annual product is about $250,000,000. In 1910 a joint board of sanitary control was organized, whose members represented employers, employees and the public, and it was given power to establish standards of hygienic conditions and enforce them. They have carried on medical examinations of employees, educated the workers in the rules of hygiene and inspected the provisions for light, safety, ventilation, cleanliness, comfort and decency.[10]

Administrative Law.[11]—Administrative law, a branch of public law, determines the form of organization, gives authority and direction to the officers, and protects both individual rights and community interests. The legislature expresses the will of the people in general outline and within the constitutional limitations, but the administration must carry out the law in detail through ordinances, regulations, orders, and personal supervision.

In both federal and state governments duties are distributed between central and local officers, and the latter are subject, in varying degrees, to superior supervision, direction and control. Cities and counties have only such powers as may be expressly conferred on them by charters which enumerate those powers, or by laws of a general scope.

In the United States there is a strong tradition unfavorable to granting administrative bodies legislative powers, and this is at times a cause of ineffective service. The executive officials are bound by the law, which often enters into many details not appropriate for general legislation. The theory is that administrators and municipalities have closely defined powers and cannot go beyond them. While the powers of the President of the United States have been enlarged, those of governors of states have been reduced. As a general rule the heads of departments are elected by the people and are not subject to the control of the governor; and, since there is no responsible executive, public business frequently lacks coherency, unity and efficiency. The "long ballot" contains so many names that the voters know very little about the qualifications of their candidates, and an election can hardly be said to express the choice of the people. One of the most serious defects of our system is the want of central power of supervision and control of local administration. County officers do much as they please, while the state board sends out circulars of advice which go unheeded. In matters of health the need of central control is so obvious that greater authority is given state boards of health, especially in times of epidemic, and here a larger measure of discretion is given to medical authorities without enumeration of their specific duties and powers. The state factory inspection is relatively independent of local influence, and

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can be made uniform, equitable and efficient in its working. The great danger is that the inspectors may be products of the "spoils system," unfit for their office, and that they may be bribed or otherwise induced to neglect the enforcement of the law. One of the grave defects of local administration and often of state administration is that it is not professional; the term of office is short and uncertain, and it is impossible to acquire skill.

Enforcement of the Law.—Usually the employers are induced to obey the law by the orders of the inspectors. If they refuse, the inspectors may bring the matter to the attention of the prosecuting attorney or the competent court, and obedience is enforced by judicial authority sanctioned by the penal provision of the statute.

Information.—One function of administration is to make records of its activity and publish information. The scientific test of the actual working of a law and of an office is the result of activity, and the evidence must be supplied by those responsible for the administration. Laboratories are properly connected with state and city offices, under professional direction, first of all to improve the service to patients and to persons exposed to danger, and in the second place for scientific investigation.

Control over the Administration.—The purpose of the mechanism of control is to promote efficiency in the service, to protect personal rights against arbitrary and illegal acts of officials, and to further social welfare. The modes of control are administrative, judicial, and legislative. The system of administration contains in its own law and regulations instructions which are designed to prevent unreasonable and injurious actions of persons armed with authority. There is also an appeal to the courts when a citizen thinks he is wronged or is in danger of being injured by persons in office. And finally the law-making assembly has large powers, upon complaint or petition, to investigate any branch of the administration and to enact laws directing and restricting the action of public servants.

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CHAPTER VI

EFFECTIVE LEGISLATION AND ADMINISTRATION

BY C. H. CROWNHART, LL. B., Madison, Wisconsin.

SECTION I

Principles of Legislation.—Legislation for safety of life and limb has come piecemeal after long experience has demonstrated its need. It has come largely as a remedy for a known evil instead of anticipating evil and preventing it. Little else could be expected from the legislature created to deal concretely with concrete things. Hence, we have legislation providing a penalty for not guarding a bull wheel, or a buzz saw, or an emery wheel; we have legislation providing a penalty for polluting a water supply in a certain way, preventing sale of dirty milk, or contaminating the air with smoke; we have legislation providing for the quarantine of certain enumerated diseases, for vaccination, or for segregation of tuberculosis patients. This kind of legislation, dealing with certain enumerated dangers to public life or health, invariably comes with much agitation and public pressure long after scientific or expert men have reached a full realization of the need of a rule of safety on the subject. The legislative rule, when expressed, often fails adequately to meet the situation, and naturally so, because the legislature meets infrequently, has many things to do, is a non-expert body, and has little time to devote to details, even though the details may be of vital importance.

For these reasons we find that factory legislation in this country has come almost solely from agitation by labor unions. Such bodies are without sufficient means to gather statistics and to vitalize facts for present action to legislatures; nevertheless, the labor unions have been impelled by shocking instances of abuse, occurring within their sight and hearing, to present convincing facts to the legislatures against effective lobbies of employers who saw the cost of a proposed change but who did not comprehend the ultimate benefits. Thus there has been very little method in this country in applying scientific study or expert advice to problems which the professional or expert man is most competent to handle.

In European countries such matter have long been dealt with through administrative orders, but with us the constitutional power to handle the subject in this manner has not been so plain. Within recent years it has begun to dawn on the public that there is really no reason why the European method is not applicable in a modified way in America. While the

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legislative power, by constitution, is given to the legislature, courts have held that the legislature may enact legislation to become effective upon the subsequent determination of a fact by another branch of the government. And the courts have held that a legislative grant of authority carries the implied power to do all things necessary to make the legislative intent fully effective. Likewise the courts have held that where the legislature delegates power to another body to ascertain a fact, courts will not set aside the findings of that body unless the findings are opposed to the the fact beyond a reasonable doubt. The method of dealing with problems requiring expert investigation was first enacted into effective statutory law in Wisconsin by the creation in 1905 of a railroad commission with adequate powers. To this commission under similar legislation was given the control of public ultilities in 1907. More or less feeble attempts were made before that time to delegate authority to various boards, but no comprehensive plan embodying the European idea of administration orders was enacted before the Wisconsin Railroad Commission Act of 1905. The Federal Interstate Commerce Commission recently has been given similar powers; railroad commissions and public utility commissions with like powers have been created in many states. As might be expected, the idea of adequately protecting and regulating property rights anteceded the more human rights of safety of life and health. But of recent years it has become more and more apparent that we must deal with the problem of life saving in a more rational way. We are gradually beginning to see what can be done by expert treatment of this purely technical subject. The methods and results of handling the health problem in the Panama Canal Zone, for example, have proved a revelation.

Wisconsin in 1911 had a vision of the future for safety legislation as applied to places of employment—the relation of master and servant, to use a common law term, or the relation of employer and employee, to use the modern expression.

Under its railroad commission regulation, the Wisconsin legislature has said that every unreasonable rate should be unlawful; then it committed to the commission the power to find as a fact the reasonable rate. With this general rule went the appropriate administrative machinery and provisions for court review. The same principle was applied in 1911 to safety in the various employments of the state. The legislature enacted the broad and comprehensive rule that every place of employment shall be reasonably safe, and safe is defined to mean freedom from danger to the life, health and comfort of employees. The legislature then committed to the industrial commission the power to find the facts as to safety. As in the case of the railroad commission, the facts are determined by orders and under the legislative scheme these orders have the force and effect of law.

Under this scheme it is plain to be seen that the legislature has laid down a rule that is good for all time. What more can be desired from a safety

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standpoint than that every place of employment shall be safe, the term "safe" including physical decency and comfort? From then on the problem takes on the aspect of scientific and expert study, as applied to each employment, to find the factors of safety and then to incorporate such factors in the proper order. Here, then, is the elastic basis of safety legislation. As science, study, or discovery may open the way to safer methods, the proper machinery is at hand to incorporate such new methods into law and make them immediately effective. The advantages of this scheme of general legislation, leaving details to be supplied by a competent body of experts, are manifest. The fundamental rule is the same all the time. Upon this basic rule the whole body of safety regulation grows day by day, year by year, discarding the dead as the living body discards dead tissue, and taking on new rules as new ideas develop safer methods. The problems of safety have become the problems of education. No longer does safety of life and limb await legislative inaction. The whole subject has become one of efficient administration.

SECTION II

Methods of Administrative Boards.—The Wisconsin commission is appointed by the governor and confirmed by the senate. The term of office is 6 years and the individual salaries equal those of the constitutional state officers. Opportunity is afforded for obtaining as assistants in the field and office efficient men and women who devote their entire time to their work.

The chief objection, raised by opponents of the administration against the methods outlined in the foregoing section, is the alleged danger of changing a democracy into a bureaucracy. Whatever the danger, in fact, it is important to the people and to the scheme itself that the administration of such a law shall be wholly consistent with the spirit of our institutions and form of government. To put it plainly, we believe in this country that the people govern and are capable of governing. The commission then must be justified on the ground that it is an agency of the people to do for the people collectively those things which it may do for them better than the people could do individually for themselves. Being a direct agency of the people, the commission should get close and keep close to the masses in order that it may correctly interpret the aspirations and ideas of the masses into progress and human welfare. The commission cannot move much in advance of public sentiment. Obnoxious rules generally cannot be enforced even though they are theoretically correct. In matters dealing so intimately with the individual and the family relations as the laws governing safety and health, education is a prime factor. And in the administration of the law the rule of safety applies: "Better safe than sorry." For this reason the law should provide, as the Wisconsin law does, for general publicity, and the commission should court the fullest publicity and coöperation. The

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term "reasonable" in the law may well be held to mean "practicable." The commission will not go far if it does not consider what is practicable, and what the people consider practicable may have quite as much bearing on the success of the law as what the commission may deem practical.

It was upon consideration of these things, and upon the further consideration that the men actively engaged in production can give most valuable advice, that the Wisconsin commission sought the coöperation of all interested persons in forming an advisory committee in working out standards of safety and health. In selecting this committee it was natural, of course, that the most progressive employers and employees should find a place. Employers made their own selection of representatives as did the laboring men. The membership of the committee was completed by representatives of the commission.

It has been an inspiration to the commission to follow the arduous work of the committee day by day and month by month. Nothing of discord, nothing but sincere, earnest effort to secure the highest practical standards has been evidenced on the part of either employee or employer. Subcommittees to deal with technical subjects have given equally valuable service. As fast as facts have developed definite conclusions, orders have been issued. Orders have been issued on the following subjects: elevators, emery wheels, machines, fire escapes, ventilation, light, laundries, electric construction, building construction, dust extractors, blowers and exhausts for fumes and gases, and many other things. Progress is being made all the time. As fast as accidents are reported, they are studied for the remedy. If bad work conditions develop, every avenue of information is sought to find relief. Employer and employee in this respect at least are in full accord. I speak in general terms; there may be exceptions (to prove the rule).

Here it is well to mention one essential difference between the administrative orders of the commission and former legislative enactment.. The legislature must necessarily speak in comprehensive terms and use the phraseology of the law. The language of such statutes often is meaningless to the business man. He may consult a lawyer, of course, but the lawyer may be as ignorant of the application of the legal rule as the business man is of the law. The commission's orders, on the contrary, are in the language of trade or business. They deal with specific things; they are illustrated when necessary with pictures or blue prints; in short, they are understandable by the man on the job. These orders are not confined to the statutes in the lawyer's office; they are published in pamphlets and mailed directly to the man interested. If the employer needs further information, the trained deputies meet him on the job for that purpose. Under the commission scheme, if an order as applied to a particular condition is found impracticable or unreasonable, the person affected may petition the commission for a hearing to be relieved from the effect of the order. This elasticity is a very valuable feature of the new idea over the old. Under the old idea mistakes could not

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be remedied until the legislature convened and many times employers were compelled to do useless things at large expense. It necessarily followed from the composition of the legislature and the time it had to devote to a technical subject that many mistakes were made; many laws were enacted which were of doubtful benefit to the workmen. Under the new method, all parties should be benefited. Changes will be made gradually. Factory conditions will improve all the time and production will not cost more but will cost less. The relations of employers and employees are improved. Safeguards do not come after legislative lobbying which is distasteful to both sides and fruitful of ill will and future friction. The whole proposition is reduced to the question of what is reasonable and practical; and when that is determined, immediate results follow, results satisfactory to all concerned.

SECTION III

Relation of Compensation to Safety.—The doctors tell us that for every violation of the laws of health, nature imposes a penalty. And we know that this is true. From the "cradle to the grave," nature is teaching us safety. Does the toddler fall? Nature tells him to be careful. Does the boy too soon connect with the green apple, or with the cucumber? Nature shouts its warning through the stomach. The barefoot boy with the turned up pantaloons leaves off his merry whistled tunes when he stubs his toe. Nature sounds its warning. Truly has it been said that "Nature is a wise old teacher."

As the young man goes out into the workshop to earn his right to live by brawn and brain, he carries with him the experience of his childhood and youth. That experience tells him to have a care, lest he experience physical pain and suffering. And day by day he gains in experience. He sees his fellow-workers fall by the wayside. He is a witness to their suffering, anguish of mind and body. He learns, too, how great is the loss to the family when the breadwinner of the family is stricken. Nature teaches him to be careful.

The news of the day shouts a thousand warnings to "be careful." But business, industry, cries "Hurry! Hurry! Hurry!—always Hurry!" The workman, the soldier of industry, is surrounded by a thousand dangers. The forces of nature, linked with wonderful machines devised by man, rise before his untrained intellect, in bewildering complexity. His sensitive nerves become callous. Too often he neglects the caution of experience and judgment, with the result that he is the victim of accident. When he is injured, he alone suffers the consequent physical and mental pain. No one can share these with him.

We know, however, and we must not allow our eyes to be closed to the fact, that neither the workman nor the employer is alone to blame for the casualties in our industries. Accidents result from many contributing causes. The great cause, the one overshadowing all others, may be called the hazard of the employment.

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By study, and by eternal vigilance on his part, the employer may lessen the hazard of industry. The employee, likewise, is capable of reducing the hazard. But the employer must not be allowed to forget that he is the captain of the industry, and with his rank goes the greater responsibility. And he must come to a realization of the fact that he cannot if he would, shift the burden from himself. The captain must select for his soldiers, competent workmen. He must assign them to the proper kind of employment, and must furnish them with safe places of employment. He must surround his workmen with fellow-servants who are competent. He must give to his employees competent foremen, proper instructions, and constant efficient supervision. He must inspect his machinery, his ways, his methods, and his safeguards at every turn. He must not only make reasonable rules for safety, but he must enforce them. And when he has done all of these things, he will still have those accidents which will come from the hazard inseparable from industry, impossible to be foreseen.

Because of the great responsibility imposed upon the employer, he, too, should accept penalty for his failures. As the sensitive nerves constantly transmit to the brain of the workman the warning to be careful, so should the sensitive nerve of business serve to warn the employer to be careful.

What is the sensitive nerve of business? I need not answer that question for you. You know that it is the dividend. You may talk about humanity until doomsday. But our feelings of compassion for the unfortunate are touched after the accident—not before. And then we spend but little time in vain regret. We turn to our business, and business means dollars.

Now, the workman is penalized in every case of accident by the suffering incident to his injury. So, too, the sensitive nerve of the employer, his dividend, must be touched in every case of accident if he is to be spurred to his full duty. This means that when a workman suffers from accident, the employer must suffer in the way most certain to teach him the care due his employee. By this method, and by this method only, can the employer be made to realize and appreciate the necessity of guarding against accident—be made to respond to his full duty in that respect. This penalty will bring response.

Just for a moment contemplate. During the 12 months from September 1, 1911, to September 1, 1912, exclusive of accidents in the railroad service, there were in the state of Wisconsin over 6500 accidents, each of which caused disability on the part of the workman for more than 7 days. This would seem to be worse than war. At least more men were injured during that time than were wounded during the same time in the Spanish-American or the Philippine wars. And mind you, 50 per cent. of the time loss incident to these casualties could have been saved by the employers had their full duty been done as here outlined.

Here then we have the relation between accident prevention and workmen's compensation. They are obviously correlated. One is the comple-

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ment of the other, and they should both be administered by the same body so that the relation of prevention and compensation may always be kept before the employer. It is for this reason that self insurance is best for safety, and next to self insurance comes mutual insurance. All insurance should carry credit for good work on the part of the employer for accident prevention, and countercharges for poor work. Let this be a contest in which money is to be saved; then watch results.

All the time the employer should be made to feel the relation of accidents to the cost of compensation. And it is workmen's compensation that brings this home to the employer as nothing else will.

We are pleased, indeed, that progressive employers everywhere are taking into account the maimed and dead workmen. They are beginning to count the cost of personal injury in dollars. This presages saving of life and limb—preserving to the family the breadwinner—preventing suffering and want.

In Wisconsin the factory inspector used to compel safety with a big stick. He was the policeman with the club. Now, the function of the commission is to point the way to safety—the employer will do the rest.

Since the workmen's compensation acts of the past 2 years, more real safety work has been done in the United States than in any 10 years preceding. This is due almost wholly to a fuller realization of the cost of accidents on the part of the employers. As the relative cost of accident becomes greater, the necessity for accident prevention becomes the more apparent.

No statute ever has been or ever can be enacted that will efficiently safeguard the worker except one that automatically provides the penalty for the employer for each and every injury received in the work places as the result of accident. The compensation act provides the penalty and at the same time subserves society as a whole by distributing the burden of loss from the weaker members of society to society as a whole. But of more importance than the relief to the injured worker, which comes from compensation, is the prevention of injury because of compensation. Heretofore the efficiency expert has studied his subject almost wholly from the standpoint of increased production to reduce the unit of cost. Let us not forget that the thing produced is only valuable as it conduces to the welfare of man. No increase of production is desirable that adds to the sum total of human misery. He is no benefactor of mankind who makes two blades of grass grow where one grew before, if in so doing he sacrifices human happiness.

SECTION IV

The Practical Equipment of the Place of Employment to Prevent Accident and Disease.—There is no definite guide for safety of life and health for the employer and employee. The careful, intelligent guarding of machines, ways and works, will work wonders. Proper lighting will greatly lessen accidents, safeguard the eyes, increase the output, increase the efficiency of

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the employee, and contribute greatly to good cheer in the institution. Properly constructed exhausts will remove dusts, gases, and impure air. Good ventilation will do much to keep the workmen fit for work. In short, mechanical devices will obviate many of the dangers to which the workmen are ordinarily exposed. The progressive employer in 6 months' time at a very reasonable outlay can make his plant up-to-date in these respects.

When he has done all these things, he has fallen far short of possible results in protecting workmen from injury and disease. Take a poor shop and equip it perfectly with all modern safeguards and you have made only about one-third the possible saving in accidents and disease. The other two-thirds must come from coöperation on the part of the workmen themselves. Education of the worker to exercise the highest degree of care is necessary. This can be done. It has been done. It is being done every day. Shop organization and discipline in the factory or workshop is just as much the duty of the employer as to build mechanical safeguards. The employer is the responsible party. He can secure coöperation of his workers, if he will. If he will take his men into his confidence, they will respond. If he will organize safety committees among the men and give them responsibility and intelligent supervision, he will get immediate returns. Accidents will decrease wonderfully and immediately. Better shop conditions will come about. He will see increased production and a healthier shop and crew. It will pay. More and better work will be done. It will not cost as much in damages for compensation. There will not be so much time lost. There will not be so much shifting of men with the consequent loss in breaking in new men.

It is not enough to make assertions. They may seem extravagant. But we have facts to prove these assertions. The new method in Wisconsin began in September, 1911, but was not fairly in operation until a year later. Here are some records, just a few out of the many, which we have of things done and being done. I compare 2 months—July and August, 1912—with the same months 1913, to show accident reductions:

SAVINGS ACHIEVED BY A FEW MANUFACTURERS COVERING TWO SEPARATE ACCIDENT PERIODS

Accidents in	July and Aug., 1912	July and Aug., 1913	Decrease, per cent.
Simmons Manufacturing Co., Kenosha	269	146	45.6
Pfister & Vogel Leather Co., Milwaukee	371	106	71.4
Nordberg Manufacturing Co., Milwaukee	177	63	64.4
Bucyrus Co., South Milwaukee	379	172	54.7
Chain Belt Co., Milwaukee	121	58	52.1
National Brake & Electric Co., Milwaukee	127	29	77.1

A still more favorable showing may be made if we compare the nature of the accidents. The time lost per accident shows up equally well. These

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figures demonstrate conclusively the possibilities in this country. We have been killing and maiming our workers by the hundreds of thousands each year. Not even the civil war compares in disastrous results in killed and wounded with the peace of industry. Industry claims its awful toll day by day until a nation is hardened to think that such is the price of industrial prosperity. The unnecessary killing of a human being is manslaughter, and the penalty for manslaughter is a term in the penitentiary. Our state prisons would be full to overflowing if the criminal law had been strictly enforced. And still you cannot indict a whole people. A people's conscience is asleep, but it is being aroused. The criminal law is not the answer. But the burden to care for the injured and their dependents is going to be charged to the industry. The employer who can save here will win in the competitive field. He can win now, and he had best put his house in order.

There are some fundamental things to do; among others I refer to the first rules of safety adopted by the industrial commission. These rules are being added to, as a study of conditions demand action. Some of the commission's orders are as follows:

Order 2.—Pulleys—guards. All pulleys over 18 in. in diameter, which are exposed to contact, must be guarded.

Order 3.—Loose pulleys. All machines, not individually motor driven, must be equipped with a loose pulley or a clutch or some other adequate means of stopping the machine quickly.

Order 4.—Belt-shifters. All loose pulleys must be furnished with a permanent belt-shifter, so located as to be within easy reach of the operator. The belt-shifter must be so constructed as to make it possible for the belt to creep from the loose pulley back on the tight pulley.

All belt-shifters must be equipped with a lock or some other efficient device which will prevent the shifter from being accidentally shifted.

Order 5.—Pulleys near shaft hanger. Pulleys must be so placed as to allow the width of the belt between two pulleys, or between the pulley and the shaft hanger, or a hook must be provided, or a guard placed adjacent to the pulley to prevent the belt from leaving the pulley.

Order 11.—Gears. All gears, where exposed to contact, must be entirely enclosed or equipped with a flange guard which must enclose the teeth of the gears. All arm or spoke gears and all web gears with holes in the web, which are over 18 in. in diameter, where exposed to contact, must be entirely enclosed.

Order 12.—Keys and keyseats. All projecting keys in shafting, where exposed to contact, must be cut off or guarded, and all keyseats in end of shafts, where exposed to contact, must be filled or guarded.

Order 9.—Flywheels. All sections of flywheels, with spokes, which are 6 ft. or less from the floor and which are exposed to contact, must be guarded.

These orders are included here only to give an idea of their practicability and to demonstrate that the commission's safety rules are expressed in language that is familiar to shop foremen and superintendents. At the present time the commission's safety orders include rules relating to public buildings, building construction, machinery safeguards, sanitation, passenger and freight elevators and electrical equipment.

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